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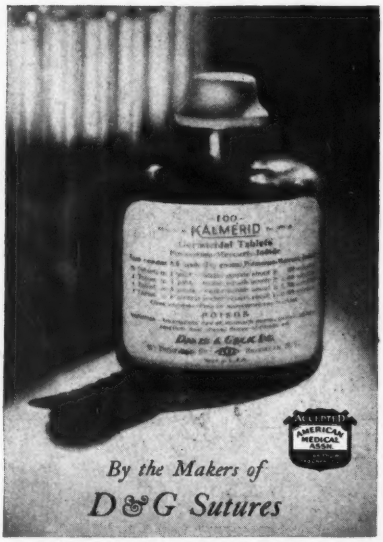

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Original Communications

A STUDY OF THE BRONCHIAL, PULMONARY, AND LYMPHATIC CIRCULATIONS OF THE LUNG UNDER VARIOUS PATHOLOGIC CONDITIONS EXPERIMENTALLY PRODUCED*

MARY E. MATHES, M.D., EMILE HOLMAN, M.D., AND FREDERICK LEET REICHERT, M.D.

SAN FRANCISCO, CALIF.

THE circulation in the lungs is unique in that a comparatively small amount of arterial blood for the nutrition of all the pulmonary structures is supplied through the bronchial artery under normal systemic pressure, and a relatively large quantity of venous blood for oxygenation is supplied through the pulmonary artery at approximately a third of the normal systemic pressure. The exact rôle played by the two vascular systems in the reaction of pulmonary tissues to disease and to pulmonary embolism has received little attention. Accordingly, we wish to present the results of a study extending over the past three years[1,2] of the two pulmonary circulations under varying pathologic conditions experimentally produced.

Method.—These studies were carried out on dogs, by injections of the vessels of the lung with Hill's mass,[3] a suspension of 20 per cent bismuth oxychloride in 11 per cent acacia which fills the vessels up to but not including the capillary bed.

Unless otherwise stated, the bronchial arterial tree was injected first, followed by the injection of the pulmonary artery. The chest was opened by splitting the sternum exactly in the midline, and with the lungs in situ the cephalad branches and the descending aorta distal to the fourth intercostal arteries were ligated. The ascending aorta was cannulized and injected, thus filling the bronchial artery. A pressure of 160 mm. Hg was maintained during this injection which was considered equal to the average systemic arterial pressure in the dog. After the injection was complete (in about fifteen minutes) the heart and lungs were removed, the trachea was cannulized, the lung was inflated to its normal size at a pressure of 10 mm. Hg and roentgenograms were made.

*From the Laboratory for Surgical Research, Stanford University Medical School, San Francisco, California.

Read at the Fourteenth Annual Meeting of the American Association for Thoracic Surgery at San Francisco, July 1, 2, 3, 1931.

Through a cannula inserted into the base of the pulmonary artery, this vessel was then injected at the normal pressure of 40 to 60 mm. Hg and additional roentgenograms were taken. In many cases the pleural lymphatic vessels were injected with India ink or mercury. The lung was preserved in formalin and after two or three days roentgenograms of the individual lobes were taken. The involved lobes were sectioned and examined grossly. Large collodion sections for microscopic study were prepared from the involved lobes and other sections were cleared by the Spalteholz-Sabin method.[4, 5] The cleared sections, when studied and dissected under low power binocular dissecting microscope illuminated by a strong transmitted light, demonstrated the intimate course and relationships of the injected vessels and lymphatics.

A. CIRCULATION IN THE NORMAL LUNG

1. *The Pulmonary Artery.*—The chief function of the pulmonary artery is to convey blood to the lung for oxygenation. It nourishes only

Fig. 1.—Animal K 2. Roentgenogram of isolated lobes of lung following injection of pulmonary artery with Hill's bismuth mass to show absence of injection beyond occlusion of artery by emboli (indicated by shots); the branches of the pulmonary artery are end-arteries and have no precapillary anastomoses.

the respiratory epithelium of the terminal alveoli. The nutrition of the bronchial walls, the lymphoid tissue, the fibrous stroma of the lung, and the walls of the pulmonary artery itself is furnished by the bronchial artery alone. Each pulmonary artery to a lobe is an "end-artery" in the sense that it has no anastomoses proximal to the capillary bed. This was definitely shown by injection of the pulmonary artery with the bismuth mass following the occlusion of one of its branches by a recent embolus (Fig. 1). A sharply outlined pyramidal-shaped area with the embolus at its apex remained entirely free of injection. If we injected a colored gelatin or saline solution which readily passes through the

capillary bed, the embolic area was colored slowly by the solution entering at its periphery, and the arteries and veins distal to the embolus were found to contain the solution. Anastomoses in the capillary bed were responsible for this injection. That the pulmonary artery is not essential to the nutrition of the lung was shown repeatedly by the relatively normal appearance of the lung, in the absence of infection, beyond an occluded branch of the artery, and also after complete ligation, at the hilum, of the pulmonary artery to a lobe.

2. *The Bronchial Artery and Vein.*—The exact distribution of the bronchial artery in the dog has been carefully described by Miller.[6] It arises in one to three small trunks from the anterior side of the descending aorta just distal to the aortic arch. Frequent variations in its origin

Fig. 2.—Animal L 24. Roentgenogram of normal lung following bismuth injection of bronchial artery showing its small size and course along the bronchi.

are met with; the artery on the right commonly arises from the first intercostal artery and occasionally from the internal mammary or subclavian arteries. It enters the lung at the hilum, where it divides to follow each bronchus as it enters the lobe. Large anastomoses connect the branches before they enter each lobe and form a coarse-meshed network around each bronchus within the lobe. The branchings of the bronchial artery are peculiar in that they leave the trunk at right angles and the branches are often as large as the trunk from which they arise. The bronchial artery extends only to the ductus alveolares where it breaks up into a capillary network. The blood of the bronchial artery is returned largely by the pulmonary vein. Near the hilum, true bronchial

veins containing valves collect blood from the bronchial arteries of that region and empty into the azygos, hemazygos or intercostal veins. Attempts to demonstrate the bronchial vein by injection have repeatedly failed. Careful dissection of the vessels showed that, unlike the pulmonary veins, they contain competent valves which prevent injection.

Roentgenograms after injection of the bronchial arterial system with bismuth oxychloride showed the delicate tracery of this artery which follows the course of the bronchus. No precapillary connections between the bronchial artery and pulmonary artery exist, as was shown repeatedly by injections of the normal lung (Fig. 2.).

If the injection of the bronchial artery was made with a colored gelatin or saline solution which passed through the capillaries, dissection of the

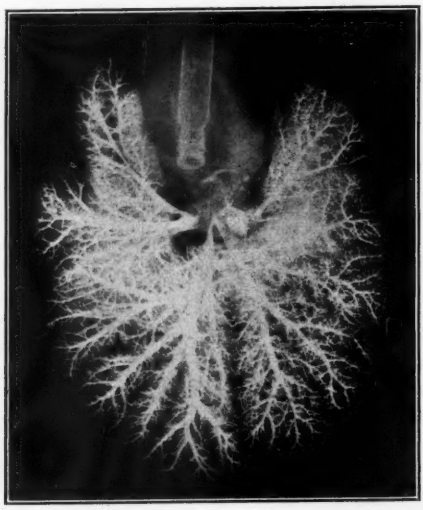

Fig. 3.—Animal L 56. Roentgenogram of normal lung following bismuth injection of pulmonary vein. The pulmonary vein has no valves and can be injected at the hilum. The bronchial vein has valves and cannot be injected.

lung showed the pulmonary artery and pulmonary vein, as well as the bronchial artery, filled with the injected solution. There is, therefore, a definite *capillary* communication between the two arterial systems.

Karsner,[7] in 1913, made the same observations and showed by a series of simultaneous injections of gelatin into the bronchial and pulmonary arteries that with the normal pressure relationships of 140 mm. Hg in the bronchial arteries and 40 mm. Hg pressure in the pulmonary artery there was practically no mixture of the blood of the two systems. Only when the pressure in the one system or the other was reduced to zero did blood flow from the one of higher pressure to the other of no pressure.

3. *The Pulmonary Vein.*—The pulmonary vein contains no valves and consequently may be demonstrated by injection of the main trunk in exactly the same manner as the artery. The appearance after injection

was similar to the artery but the vein ran a slightly different course (Fig. 3). The vein corresponds to the pulmonary artery in presenting no anastomoses between its larger branches, although there are capillary anastomoses. This was easily demonstrated by ligation of the main vein

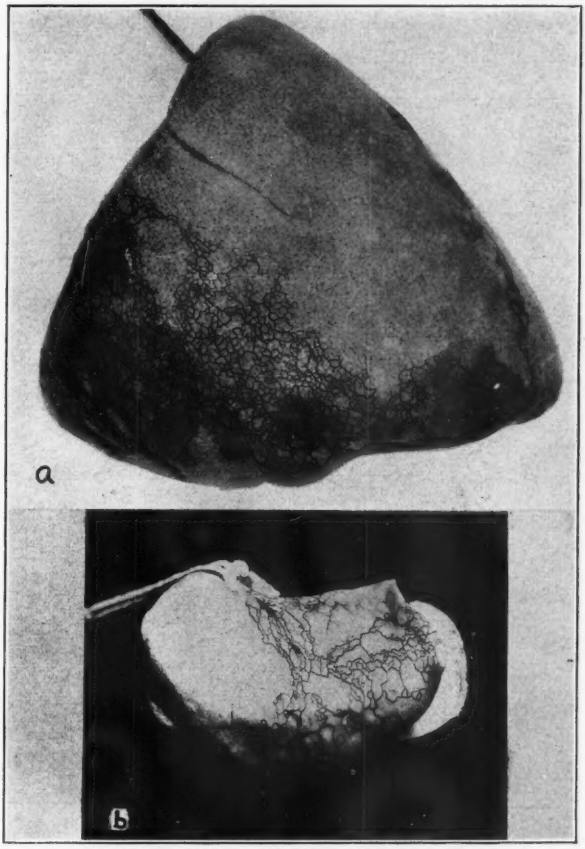

Fig. 4. Animal K 17. (a) Photograph of normal lower lobe of lung following India ink injection of visceral pleural lymphatics. (b) Photograph showing pleural lymphatic drainage converging toward the hilum. The pleural lymphatics may dip into the pulmonary tissue anywhere to enter larger lymphatic vessels accompanying the bronchi to the hilum.

at the hilum of a lobe distal to a small branch of the vein. Injection of the pulmonary vein with bismuth showed the segment of lung beyond the small unligated branch completely filled without any injection of the lung distal to the ligated vein.

4. *The Lymphatics.*—The lymphatics of a normal lung vary markedly in size. They are seen as thin-walled, delicate, transparent vessels just beneath the visceral pleura. Often single loops of these vessels are enlarged and plainly visible, and in some instances a more extensive net-

work appears. Injections of India ink, made with a fine needle and Luer syringe, directly into one of the larger lymph vessels brought out the lymphatic pattern. The injection was stopped by valves at irregular intervals. The lymphatic vessels form a freely anastomosing fine network just beneath the pleura (Fig. 4). During injection some vessels were seen emerging along the large vessels or bronchus at the hilum to enter a peribronchial lymph node. In cleared specimens branches from

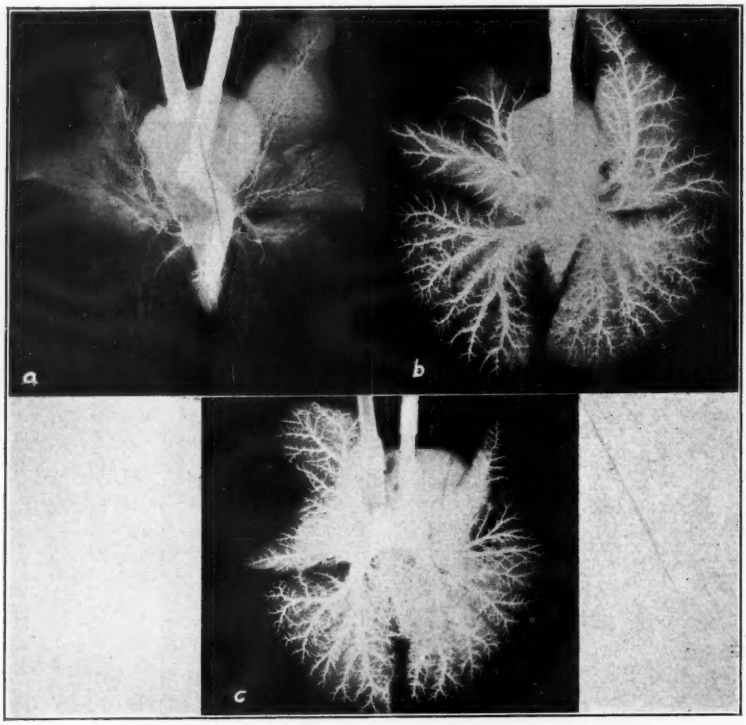

Fig. 5.—Animal L 53. Roentgenogram of lung showing bronchopneumonia in both upper lobes accompanied by (a) generalized dilatation of bronchial artery to the affected lobes; (b) incomplete filling of peripheral branches of pulmonary vein; and (c) incomplete filling of peripheral branches of pulmonary artery due either to capillary and precapillary thrombosis or to closure of vessels by compression from surrounding edema of tissues.

the pleural lymph vessels were seen running into the substance of the lung along either a vein, artery, or bronchiole, and formed a meshwork around the vessel or bronchus, terminating in a peribronchial lymph node in the hilar region.

B. CIRCULATION IN (1) PNEUMONIA; (2) ATELECTASIS

The material for the study of the circulation in bronchopneumonia was obtained largely from stock dogs suffering from distemper. Some of

these animals died of bronchopneumonia, others were sacrificed in earlier phases of the disease. Distemper begins with an acute sinusitis and upper respiratory infection which is followed by bronchopneumonia varying in extent from areas of patchy consolidation to a massive consolidation of the entire lung.

All of the lungs injected in this series showed definite constant features. The most striking feature was a generalized moderate dilatation of branches of the bronchial artery supplying the consolidated area (Fig. 5, *a*). These vessels never showed the extreme increase in size of the bronchial artery seen after the occlusion of a branch of the pulmonary artery by embolism or ligation.

Bismuth injection in 54 lungs containing areas of pneumonic consolidation, 26 of which showed massive consolidation, demonstrated in every case a moderate but definite dilatation of the bronchial arteries of the affected region. In no one of these 54 cases did the bismuth from the bronchial vessels pass into the pulmonary artery or vein. This is understood when it is remembered that approximately normal pressure relationships between the two systems existed (Karsner[7]). Blue gelatin or saline solution injected into the bronchial arterial system filled the pulmonary artery much more rapidly and completely in the area of consolidation than in the normal parts of the same lung. This suggests definite dilatation of the capillary anastomoses between the two systems in the inflammatory area but not of sufficient degree to allow the passage of bismuth particles.

The dilatation of the bronchial artery becomes evident after two to four days, its extent depending upon the severity of the inflammatory process. In the presence even of a pale, meaty consolidation (of three to six weeks' duration), the bronchial artery presented the same moderate dilatation.

The pulmonary artery was injected in 50 cases of pneumonic consolidation. The appearance upon casual examination differed little from the normal, but careful examination revealed characteristic changes (Fig. 5, *c*). The main trunks were well filled but the fine terminal arterioles were poorly injected and had throughout the consolidated areas a broad blunted appearance instead of the normal delicate outline. Thromboses in many of the capillaries did not allow free escape of fluid blood ahead of the injection mass; consequently the bismuth did not penetrate the smaller arterioles and a blunted appearance was the result. Colored saline solution injected into the pulmonary artery under normal pressure returned by way of the pulmonary vein but colored the consolidated area in a blotchy manner when compared with the even coloring of the normal lung. Many of the capillaries remained patent and even dilated although not sufficiently dilated to allow the bismuth suspension to pass, whereas others were definitely occluded by fibrin or clot. These changes were not apparent in the earliest stages of bronchopneumonia but were definitely present after the first two or three days.

The pulmonary vein gave the same picture after the injection of bismuth as the pulmonary artery (Fig. 5, *b*). The vein was more difficult to inject because of postmortem clots which occluded large vessels thus producing artefacts, but sufficient injections were made to assure us that there was no true occlusion of large venous branches and only capillary disturbances occurred.

Lymphatics.—In the earlier stages of consolidation during the edematous and congestive phases, the visceral pleural lymphatics were uniformly dilated and often contained blood-tinged fluid. Injection of these surface vessels brought out an extensive pleural network. Some of the vessels dipped abruptly into the pulmonary tissue and followed

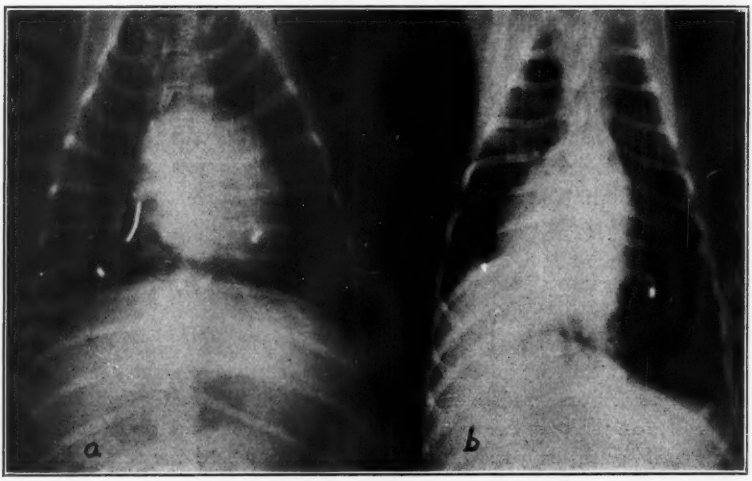

Fig. 6.—Animal K 80. Roentgenograms of chest (a) immediately after introduction of plug of sea sponge into bronchus of right lower lobe; (b) three days later, showing atelectasis of this lobe, elevated diaphragm and shifting of cardiac shadow to right. Animals in which the primary bronchus of a lobe was permanently occluded by a sea sponge invariably died due to infection and necrosis beyond the obstruction.

the vessels to the hilum where they entered an enlarged lymph node. The peribronchial nodes, as well as the substernal nodes, were enlarged and edematous.

2. ATELECTASIS

Septic and aseptic. Atelectasis of one lobe of the lung was produced by a tightly rolled, dried sea-sponge introduced into the primary bronchus through a bronchoscope. After the sponge was in place it was moistened with a few cubic centimeters of water and packed tightly into the bronchus to prevent its expulsion by coughing. Immediately after occlusion of a bronchus by a sea-sponge, absorption of the alveolar air occurred and rendered the lobe airless after a few hours. After twenty-four hours the typical signs of collapse of the plugged lobe were evident (Fig. 6). The chest on the side of the bronchial occlusion had a

smaller capacity, the diaphragm was elevated, and the heart and mediastinum were displaced markedly toward the side of the collapse. The animals showed a severe systemic reaction, with fever, rapid respiration and cough, *invariably followed by death unless the plug was displaced.* Of 10 normal animals receiving a plug of sea-sponge in the bronchus of one lobe, 5 died during the first three days, 2 lived seven days, and the other three lived ten days, eighteen days, and twenty-three days, respectively.

When the chest was opened at necropsy, 26 to 48 hours after the occlusion of a bronchus, the involved lobe appeared larger than the surrounding lung which had collapsed on opening the chest. But when the intrabronchial pressure was raised to the normal level of 10 mm. Hg so that the lung just filled the thoracic cage, the atelectatic lobe was considerably smaller than the normally inflated lobe. As previously pointed out (1) failure to inflate the lung to the size that will exactly fill the thoracic cage accounts for the contradictions in published reports on the relative size of atelectatic, consolidated, and normal lungs.

After 36 to 48 hours the atelectatic lung which was previously quite small, firm, and beefy red, showed definite inflammatory changes; swelling, edema, and areas of necrosis appeared. A clear, pleural effusion usually accompanied this stage. Six animals in our series showed areas of gangrene distal to the bronchial plug.

To determine the effect of embolism and bronchial obstruction in the same lobe, in certain animals a sterile embolus was introduced in the jugular vein, and after noting its position in the lung under the fluoroscope, the primary bronchus to the embolized lobe was also occluded with a sea-sponge. When embolism and bronchial obstruction were produced simultaneously the subsequent course of events differed not at all from that occurring after bronchial obstruction alone; all animals died of infection and necrosis of pulmonary tissue beyond the bronchial obstruction. If, however, the embolism was produced first and bronchial obstruction four weeks later, the animal survived, presumably due to the effect of an increased filling of the bronchial artery because of dilatation brought about by the embolism. As will be described later, embolism of the pulmonary artery invariably results in dilatation of the bronchial artery and therefore in increased nutrition to the lobe. A similar effect could be produced by ligation of the pulmonary artery at the hilum. This too leads to bronchial artery dilatation and if after ten days the bronchus to the same lobe was occluded by a sea-sponge, death did not occur. This was in marked contrast to the fact that death due to infection and gangrene had occurred in ten other animals following complete bronchial obstruction by a sea-sponge.

It só happened that two sterile emboli were introduced in two cases in two different lobes, followed by bronchial obstruction in one of the embolized lobes, but not in the second. However, an abscess also occur-

red in the lobe containing the sterile embolus only—undoubtedly due to secondary infection and necrosis in the atelectatic lobe, a phenomenon already described.[1]

In all of the atelectatic lungs injection of the bronchial artery showed it to be not only patent but moderately dilated as in bronchopneumonia, even in a gangrenous lobe (Fig. 7, *a*).

The pulmonary artery filled well in the atelectatic lung and during the first twenty-four hours often could not be distinguished from the normal except by the tortuosity of the vessels resulting from the collapse of the lobe. As the inflammatory process progressed the pulmonary artery presented the appearance seen in a pneumonic consolidation, the terminal branches being poorly filled and characteristically blunted (Fig. 7, *b*). Inflammatory changes produced occlusions in some of the

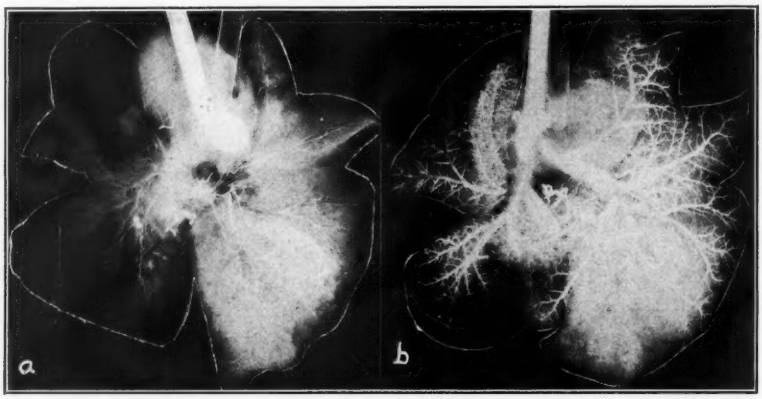

Fig. 7.—Animal K 80. (a) Roentgenogram of lung following injection of bronchial artery seven days after bronchial occlusion of right lower lobe by sea sponge. Marked parenchymatous necrosis throughout this lobe accompanied by dilatation of bronchial artery; (b) after pulmonary arterial injection.

capillaries while others remained intact and dilated. We were unable to observe the late effects of simple atelectasis produced by the sea-sponge as in every instance fatal infection occurred. A clinical application of this observation suggests that unless a bronchial plug causing massive atelectasis is promptly displaced severe and probably fatal inflammatory changes may occur. Presumably the complete occlusion of a bronchus prevents free drainage from that lobe and consequently infection which has been introduced from the mouth and throat may thrive in the stagnated lobe.

Lymphatics.—The lymph nodes of the chest and the peribronchial nodes of the atelectatic lobe are enlarged after twenty-four to forty-eight hours. The pleural and deep lymphatics are diffusely dilated as in pneumonia.

Sterile Atelectasis.—When the bronchus of a normal lung was divided aseptically by operation and its ends inverted, the lobe became atelectatic,

small, red, and meaty in appearance and remained so as long as observed (two months). There was no systemic reaction and the upper lobe almost completely filled that half of the chest. At necropsy the atelectatic lobe was small, completely airless and entirely without evidence of infection. The bronchus was filled with mucus and was slightly dilated. The pulmonary artery was patent but tortuous due to collapse of the lung tissue, the bronchial artery was of normal size. After atelectasis of two months' duration there was a moderate amount of fibrosis. The atelectatic lobe could be partially inflated by a cannula introduced into its bronchus, but it attained only half the normal size of the lobe.

Contrasting the atelectasis produced aseptically as in the above experiment with that produced by a plug of sea-sponge, we find that the different effects are due entirely to the factor of infection as the double blood supply is in both cases intact and adequate.

C. CIRCULATION IN EMBOLISM

General Considerations.—The occlusion of a branch of the pulmonary artery by an embolus abruptly arrests the flow of non-aerated blood in a pyramidal-shaped area of the lung distal to the site of the embolus. The pulmonary arterial pressure of 20-40 mm. Hg is suddenly reduced to zero in the area distal to the embolus. The pulmonary capillary anastomoses between the vessels of the embolic area and the adjacent pulmonary vessels furnish a small amount of blood to the area but at a very low pressure.

The bronchial arterial supply to the embolic area remains undisturbed but the pressure relations between the two systems are altered. The bronchial arterial pressure remains at normal systemic level while the pulmonary arterial pressure approaches zero. As a result, arterial blood enters the pulmonary vessels through the bronchial artery, which dilates in response to the increased flow through it. The bronchial arterial dilatation is permanent and the embolic area, in the absence of infection, remains well-nourished and is of normal appearance, although the function of gaseous exchange in this segment of the lung is eliminated. The pulmonary vein in the embolic area is patent and can be filled with bismuth by injection at pressure of 10 mm. Hg.

Sterile Embolus.—Sterile emboli were introduced according to the method suggested and first used by Holman[8,9] for the production of pulmonary abscesses. The first embolus was introduced into the proximal end of the jugular vein and forced along with saline solution, the animal lying on its side. After about five minutes the animal was turned to the opposite side and the second embolus introduced. This procedure influenced the course of the emboli so as to place them in opposite lung fields. The size of the embolus was constant, causing, usually, occlusion of a third degree artery.

The systemic reaction following the introduction of two sterile emboli into the pulmonary circulation was mild. The rise in temperature was

no more than one degree centigrade during the first two days after the operation. The pulse and respiration often showed a slight temporary increase. Examination of the lungs of dogs sacrificed at varying intervals from twenty-four hours to ten months subsequently revealed extremely slight changes in the parenchyma of the embolic area. During the first four days there was evidence of a mild congestion and in some

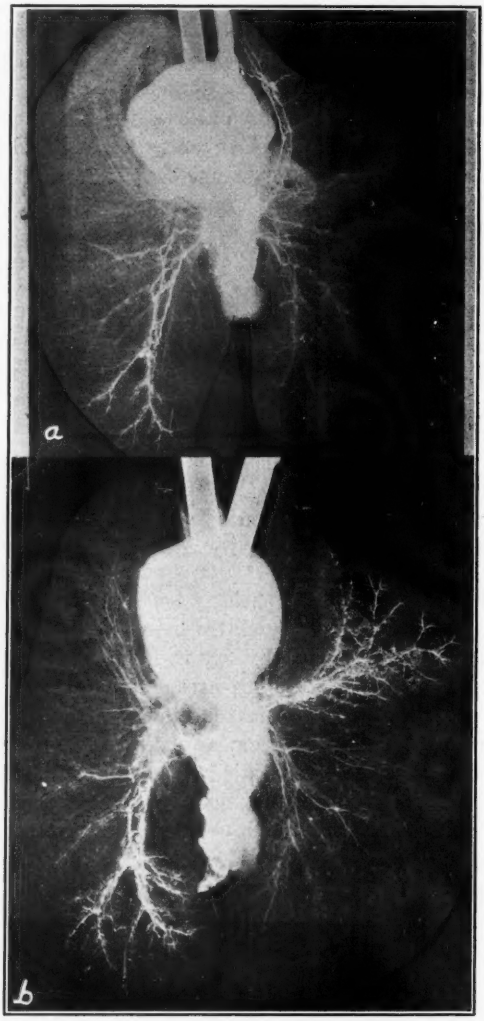

Fig. 8.—(a) Animal K 38. Roentgenogram of lung one month after two sterile emboli had been introduced into jugular vein showing definite dilatation of bronchial artery leading to embolized area. (b) Animal K 40. Roentgenogram ten and one-half months after two sterile emboli had been introduced into pulmonary artery showing permanent dilatation of the bronchial artery and injection of the pulmonary arterial branches in the embolized area through the bronchial artery.

cases a slight extravasation of blood into the alveoli but not sufficient to be demonstrated in the roentgenograms of the chest or of the excised lung. After this time the embolic area was characterized only by its slightly paler grayish color. The area often could not be located in the deflated lung but after the normal intraalveolar pressure of about 10 mm. Hg was established, the delicate but definite color change became apparent at once.

The pulmonary artery was completely and permanently occluded by the embolus. A small amount of blood flowed into the embolic area by way of capillary anastomoses from the adjacent pulmonary arterial branches, but the circulation of the embolized area was practically entirely taken over by the bronchial artery which in all cases was definitely dilated (Fig. 8, *a* and *b*). The dilatation became apparent two to four days after the embolism and increased during the first eight to fourteen days. After this time it remained relatively constant. The following protocol illustrates the procedure by which the vessels were filled after sterile emboli had been introduced.

Animal K 127. On April 10, 1930, two sterile emboli were introduced into the pulmonary circulation by way of the jugular vein. The animal was lively and well following this procedure and was killed after eight days. Necropsy revealed a grossly normal appearing lung. The embolic areas were located only after careful observation. One was in the right lower lobe, the other was in the right middle lobe. While the lung remained in situ the pulmonary artery was injected with warm normal saline solution colored with Berlin blue at a pressure of 30 mm. Hg. The entire lung was colored with the blue almost immediately except for the two embolic areas which became sharply outlined. After two minutes the embolic areas began to show a blue color in the vessels about the periphery and after ten minutes the entire area was blue but the color was not as intense as that of the surrounding lung. The bronchial artery was then injected with saline solution colored with Congo Red. The embolic areas immediately changed to a purple color whereas the rest of the lung became purplish only after a few minutes. The pulmonary artery was injected with bismuth after removing the lung from the chest, and roentgenograms subsequently showed complete occlusion of two pulmonary arterial branches by the emboli.

Animals killed three months and ten months following the introduction of sterile emboli showed grossly normal lungs in which the embolic areas were located only after careful inspection. On section of the lobe the pulmonary artery was found to be entirely occluded and the embolus was surrounded by a capsule of fibrous tissue. The lung tissue in the embolized area was indistinguishable from the normal.

The blood supply was established either by anastomoses between adjacent pulmonary arterial capillaries which in a few cases were dilated sufficiently to allow the passage of bismuth particles or by dilatation of the bronchial artery and of its anastomoses with the pulmonary artery, the second method being the one more frequently observed (Fig. 9).

Lymphatics.—The presence of a small sterile embolus produces no change in the lymphatics of the lobe as far as we have been able to determine.

Large Sterile Emboli.—Sterile emboli of sufficient size to block the pulmonary artery which supplies half of one lobe of the lung produced the same type of reaction, though more pronounced, as a smaller embolus There was a greater rise in temperature, pulse, and respiration. The lungs showed more congestion in the earlier phase and a moderate fibrosis in the later stages. There were fewer pulmonary arterial anastomoses, the dilatation of the bronchial artery was greater, and the bronchopulmonary arterial anastomoses were more evident.

Infected Embolus.—Pulmonary embolism complicated by infection produced marked changes in the lung. Infection occurred in several different ways: 1. The infection of the embolic area may arise from the

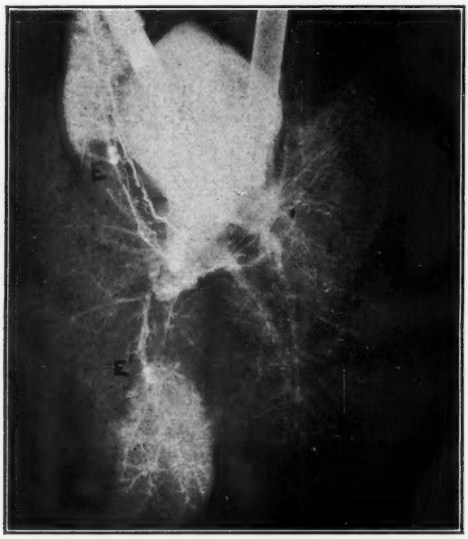

Fig. 9.—Animal K 65. Roentgenogram of lung forty-five days after introduction of two sterile emboli. Injection of bronchial artery resulted in complete filling of pulmonary artery in the embolized areas beyond embolus (E and E').

embolus itself, that is, experimentally, from an infected jugular embolus, or, clinically, from an infected blood clot arising in an operative field containing bacteria.

2. The embolic area produced by a sterile embolus may become infected (a) by a bacteriemia present when the sterile embolus was liberated[1]; (b) by a bacteriemia arising subsequent to the liberation of the sterile embolus[1]; (c) by direct invasion of the sterile embolic area by bacteria from the trachea and bronchus (Van Allen[10]).

The changes in the parenchyma of the lung resulting from such emboli, by whatever means they may have become infected, varied remarkably. Transient pneumonitis, local consolidation, abscess formation, and in a few cases hemorrhagic consolidation of the entire lobe were all observed

(Fig. 10, *a*), and occasionally gangrene of the entire embolized area was found.

It is interesting to record here the experiences of Virchow,[11] who found that rubber emboli introduced into the jugular vein never produced any pulmonary changes, but when he employed bits of muscle from the neck he was startled to find circumscribed inflammation and even abscesses of the lung. He, of course, was working without a knowledge of microbic infection, and no measures were taken to prevent infection of the bits of muscle which he introduced into the jugular vein. They, therefore, acted as infected emboli. These older experimenters, Virchow, Cohnheim,[12] et al., in attempting to produce hemorrhagic infarction of

Fig. 10.—(a) Animal K 64. Roentgenogram of lung showing consolidation of left lower lobe of two and one-half days' duration accompanied by prompt dilatation of bronchial artery. (b) Animal K 25. Roentgenogram of lung thirty-one days after introduction of infected pulmonary arterial embolus showing abscess cavity with shrinkage of lobe and markedly enlarged and tortuous bronchial artery. This dilatation of bronchial artery in the presence of pulmonary infection is undoubtedly an important factor in the control of infection and in the repair of injured pulmonary tissues.

the lung were surprised to find such varying results following the introduction of emboli into the lung, but this marked variation can probably be entirely explained on their ignorance of the pyogenic organisms, and on the fact that some of their emboli were infected, while others were practically sterile. Bland emboli such as paraffin and rubber would not produce nearly the same effect even though infected, as would a bit of infected organic material such as muscle.

An important factor in the production of these varied changes was the development of the collateral arterial supply which was derived chiefly from the bronchial artery. The vascular changes which followed an embolus complicated by infection were of the same nature as those seen after a simple sterile embolus but they were more marked and progressed with greater rapidity.

Forty-eight hours after the introduction of an infected embolus a definite shadow indicated the embolic area in the roentgenograms made either of the chest or of the excised lung. Dilatation of the bronchial artery was definite in the injected specimen of animals sacrificed at this time (Fig. 10, *a*). The bronchial artery supplying the portion of the lobe in which the embolus was located became progressively enlarged until after ten to fourteen days it was very tortuous and many times its normal size. It was easily seen as it followed the course of the bronchus and as it formed a network around the embolus itself. Many large branches freely communicating with each other entered the embolic area by passing along the bronchi and bronchioles. When an abscess cavity

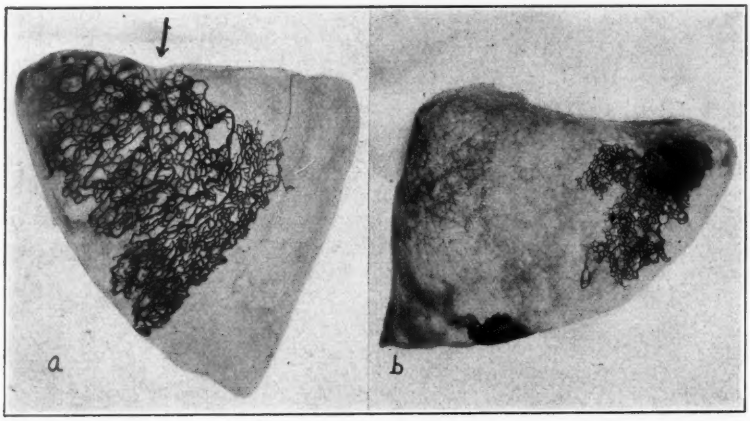

Fig. 11.—Animal K 2. (a) Photograph of lung following India ink injection of lymphatics showing dilated pleural lymphatics in left lower lobe containing an abscess (indicated by arrow); (b) pleural lymphatics of normal right lower lobe.

was present the dilated branches of the bronchial artery were found traversing the wall of the abscess (Fig. 10, *b*).

Two factors operated in this dilatation of the bronchial artery. As in sterile embolism the lowering of the pulmonary arterial pressure to zero by the embolus allowed an increased flow through the bronchial vessels. Secondly the presence of infection and active inflammation added a toxic dilatation of the bronchial artery so that the enlargement was greater and more rapid than that seen in the presence of a sterile embolus. It was observed also that the pulmonary artery beyond the embolus very soon became filled through injection of the bronchial artery. In some cases quite a complete pulmonary arterial pattern could be traced. The time necessary for this dilatation of the small communications between the bronchial and pulmonary arteries varied considerably. The earliest demonstrable filling of the pulmonary artery by way of the bronchial artery was eight days after the embolism.

Injection of the pulmonary artery of a lung containing an infected embolus showed a normal distribution and appearance of the bismuth up to the site of the embolus. Beyond the embolus lay a wedge-shaped uninjected area of lung. In a number of cases, and especially after the elapse of a considerable period of time (three months to one year), the pulmonary vessels in the embolic area were partially filled with bismuth by means of anastomoses with the adjacent pulmonary arterial branches. The pulmonary vein in the embolic area was patent.

It is apparent from the above studies that after embolism the blood supply in the embolic area was reestablished by two routes—first by a dilatation of the bronchial artery which furnished arterial blood under a relatively high pressure, secondly by dilatation of anastomoses in the pulmonary capillary bed, which supplied nonaerated blood at a very low pressure. The first source we believe to be the predominant one.

Lymphatics.—In a few cases the subpleural lymphatics have been definitely dilated in the lobe containing the abscess (Fig. 11). Cleared sections in these cases show the lymphatic vessels following the artery, vein and bronchus to the hilum. In the majority of cases no definite relationships could be traced between the lymphatics and the abscess except in the early congestive phases. At this time the lymphatics were prominent over the surface of the affected lobe and often contained blood-tinged fluid.

D. CIRCULATION AFTER ATTEMPTED LIGATION OF THE BRONCHIAL ARTERY TO ONE LOBE

Method.—All of the operations for ligations of vessels of the lung were done under intratracheal ether anesthesia preceded by morphine. The incision was made through the sixth interspace in the left chest and the hilum was exposed by spreading the ribs. During the closure the lung was inflated completely to avoid a complicating pneumothorax. The wounds healed per primam and most of the animals recovered rapidly from the operation. Their progress was followed and frequent roentgenograms of the chest were taken. After given lengths of time they were sacrificed and the lungs injected and studied as before described.

The isolation and ligation of the entire bronchial artery was found to be impossible after several attempts. The main arteries arising from the aorta were tied but in each case at autopsy the lung was entirely normal and the bronchial artery injected in its entirety from bronchial vessels arising from the intercostal artery.

Interruption of the bronchial arteries supplying one of the lower lobes was attempted by stripping all of the loose areolar tissue from around the pulmonary artery, the pulmonary vein and the bronchus. At autopsy the lobe was normal and injection showed branches of the bronchial artery remaining in the submucosa of the bronchus.

Due to their multiple origins, free anastomoses, and deep situation as they run from the anterior surface of the aorta to the hilum of the lung and within the walls of the bronchus, any mechanical disturbance in the bronchial arterial circulation is probably a very rare clinical occurrence. Embolism would be most improbable, thrombosis is unlikely and would be compensated for by collaterals, and any mechanical injury sufficient to cut off the bronchial artery would undoubtedly also cause serious and even fatal injuries to other structures.

E. CIRCULATION AFTER LIGATION OF THE PULMONARY ARTERY TO ONE LOBE

The pulmonary artery could be completely ligated at the hilum of one lobe of the lung without affecting the viability of that lobe if strict asepsis was maintained. The lobe which was then supplied by the bronchial artery alone showed only a moderate amount of fibrosis. No abscess formation, consolidation, or necrosis occurred unless infection was introduced through some route such as the blood stream, lymphatics, or bronchus.

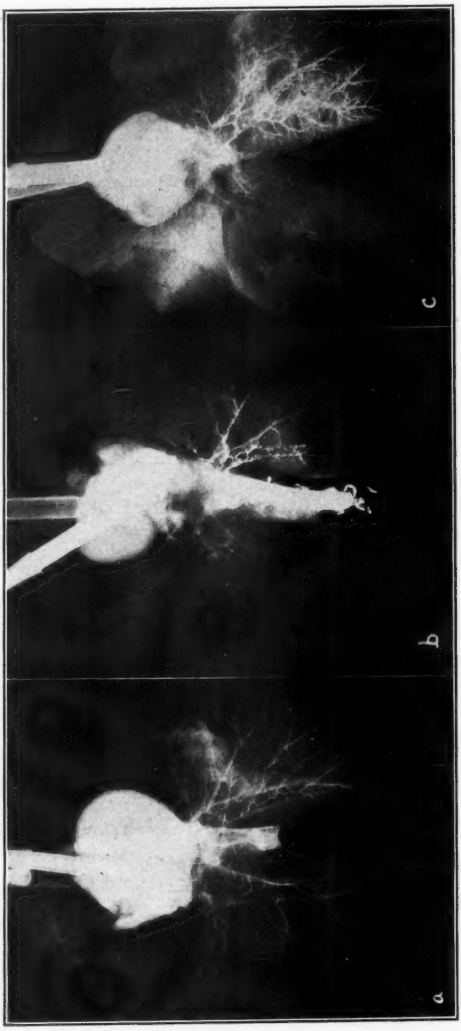

Fig. 12.—(a) Animal K 114. Roentgenogram of lung seven days after ligation of pulmonary artery to left lower lobe showing slight shrinkage of the lobe and slight dilatation of bronchial artery. (b) Animal K 106. Sixteen days after ligation of pulmonary artery to left lower lobe with moderate shrinkage of lobe and marked dilatation of bronchial artery. (c) Animal K 90. Twenty-five days after ligation of pulmonary artery to left lower lobe showing moderate shrinkage of lobe, with marked dilatation of bronchial artery and almost complete filling of pulmonary artery through the bronchial artery.

Complications causing necrosis in the lobe were frequent in ligation experiments but in each case we were able to prove a definite cause. In four out of twelve experiments in which the pulmonary artery to one lobe was ligated, there was destruction of tissue in the lobe. In one of these animals the infection arose from a deep suppurating bite on the face present before operation (Fig. 12, *c*). In a second the lobe was compressed by a hemothorax following operation, a third animal had distemper before operation. The fourth had a collapse of the upper lobes, as well as of the lower lobe the pulmonary artery of which was ligated, caused by a thick plug of mucus which was found to occlude completely the main bronchus to that lung.

Twenty-four hours after the pulmonary artery was ligated, the affected lobe was slightly smaller than normal and was somewhat pale, although otherwise it appeared normal. Injection of the bronchial artery gave a normal outline of that system. Seven days after ligation of the pulmonary artery the lobe was about three-fourths of its normal size, it was air-containing, and was without hemorrhage or consolidation. At this time the bronchial artery to the affected lobe was moderately dilated (Fig. 12, *a*). After sixteen days the lobe remained moderately shrunken but was air-containing and slightly congested without extravasation of blood, and the bronchial artery was markedly dilated (Fig. 12, *b*). The pericardiophrenic artery and the mammary artery on the side of the ligation showed slight dilatation and branches could be seen entering the lung. The esophageal arteries were enlarged with branches following along the interlobar septum into the lung. The first three intercostal arteries were dilated and branches passed through adhesions in the region of the operative scar into the lobe of the lung. In twenty-five days the above changes were more marked and there was considerable fibrosis and shrinkage of the lobe (Fig. 12, *c*). The bronchial artery was markedly dilated, and bismuth suspension injected through it entered the pulmonary branches and filled them up to the larger trunks. A considerable collateral supply from the intercostal arteries entered the lobe through the adhesions of the visceral pleura to the chest wall in the region of the operative scar. Two months following ligation of the pulmonary artery the lobe still contained air but was pale and moderately fibrous and about two-thirds the size of the normal lobe. The bronchial arterial dilatation was marked. Injection of the bronchial arterial system also filled the pulmonary artery of that lobe quite completely to a point just distal to the ligature about the artery. The pleural adhesions contained numerous small arteries running into the ligated lobe from the intercostal arteries.

The pulmonary artery was injected in each of these cases and found to be completely occluded at the hilum by the ligature. Ligation at the hilum, of course, eliminated the possibility of a collateral blood sup-

ply by way of anastomoses from the pulmonary artery itself, thus leaving the bronchial artery as the only afferent vessel to the lobe.

In embolism of a branch of the pulmonary artery to a lobe there was some flow of blood through the capillary anastomoses of the pulmonary artery itself (K-127), but blood from the bronchial artery also entered the pulmonary artery, although not so readily as when the pressure in the pulmonary artery was absolutely zero as was the case after the main artery had been ligated. The exact location of the anastomoses between the pulmonary and bronchial arterial systems could not be discovered either grossly or in the roentgenograms of injected specimens. Serial sections 3 to 4 mm. in thickness through the entire injected lobe, made transparent by the Spalteholz clearing method,[4] were studied with a low power binocular dissecting microscope using transmitted light. This revealed the relationships of the vessels. No connections between the two systems could be found in the peripheral capillary bed. The anastomosis occurred as a short trunk connecting a moderate-sized pulmonary vessel with a bronchial arterial trunk at a point where the two ran close together. These anastomoses were not numerous, not more than two or three were found along each pulmonary arterial branch of the third degree.

F. CIRCULATION AFTER LIGATION OF THE PULMONARY VEIN TO ONE LOBE

The complete ligation of the pulmonary vein to one lobe of the lung usually though not invariably caused the death of the animal in four to five days. The pleural cavity was filled with a thin, noncoagulable, blood-stained effusion. The lobe was large, very firm, and of a deep red, liver-like consistency. The lymphatics over the surface of the lobe were numerous and engorged with blood-stained fluid. The hilar and mediastinal lymph nodes on the affected side were large and filled with lymph. The bronchial artery was of normal size and distribution. The pulmonary artery in the lobe whose vein was ligated was injected poorly in its peripheral portion because of the venous stasis. A cut section of such a lobe showed the veins engorged and distended with blood.

If the ligation of the vein was not complete the animal survived and the changes in the lung were slight. In a few cases a small aberrant branch from the vein of the left upper lobe entered the lower lobe over the posterior surface of the pulmonary artery. This small vein was sufficient to prevent complete stasis after ligation of the main vein. As a result the lobe became only moderately congested and the pleural cavity contained only a very small amount of thin noncoagulable fluid. Injection of the venous system with bismuth suspension showed that the small vein supplied a limited area around the hilum. A little of the bismuth passed through the venous capillary bed and was found in the large veins of the lobe.

G. CIRCULATION AFTER LIGATION OF THE PULMONARY ARTERY AND VEIN TO ONE LOBE

Both the pulmonary artery and vein to a lobe of the lung were completely ligated at the hilum without causing the death of the animal. The lobe was maintained in a fair state of nutrition by the bronchial artery and vein and their collaterals. The changes were more marked than after the ligation of the pulmonary artery alone. The lobe became fibrous and shrunken to about half normal size, but unless infection entered there was no hemorrhage or necrosis and the lobe remained partially air-containing.

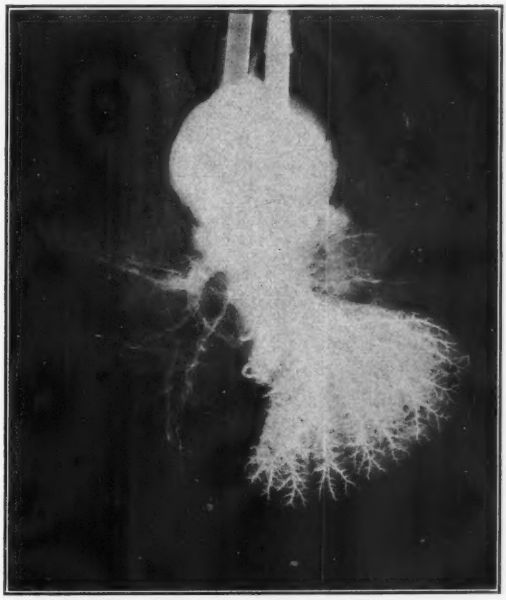

Fig. 13.—Animal K 99. Roentgenogram following bismuth injection of bronchial artery two months after ligation of pulmonary artery and vein to left lower lobe showing complete injection of pulmonary artery to the lobe by way of the bronchial artery.

The collateral blood supply was more rapidly and extensively developed after ligation of both artery and vein than after ligation of the artery alone. The bronchial artery was greatly dilated and received additional blood by way of dilated branches from the esophageal vessels which pass along the median septum into the lobe from the pericardiophrenic artery and other branches of the internal mammary artery as well as branches of the intercostal arteries running through the pleural adhesions. The dilatation of the bronchial artery became evident after the second day, and after seventeen days bismuth injection through it entered the pulmonary artery of that lobe. After one month

the pulmonary artery of the lobe could be completely injected with bismuth through the bronchial artery (Fig. 13).

The pulmonary artery and vein to the left lower lobe were ligated in seven dogs. Five of them recovered readily and suffered no ill effects from the operation (Fig. 14). The remaining two developed fever and rapid respiration, one had a wound infection and necropsy twenty-one days later showed a large multilocular abscess in the lobe to which the artery and vein had been ligated. The other dog had a reddened throat with numerous small ulcerations which were noted at operation. At necropsy seven days later, the lobe whose artery and vein were ligated was gangrenous.

Fig.14.—Animal K 93. Photograph of lung showing normal appearance and only slight shrinkage of right lower lobe twenty months after ligation of artery and vein to this lobe. As in Fig. 13, the injection of the bronchial artery to this ligated lobe resulted in complete injection of the pulmonary artery.

Searching through the cleared sections of the injected lobes short anastomotic vessels could be found connecting the bronchial artery with the pulmonary artery. These connections invariably occurred between a bronchial vessel about 1 mm. in diameter and a pulmonary vessel around 2 mm. in diameter.

SUMMARY

1. Blood reaches the lungs through two circulatory systems: (a) the bronchial artery supplies nutrition at systemic pressure; (b) the

pulmonary artery supplies blood for oxygenation at a pressure approximately one-third of that in the bronchial artery.

2. The bronchial artery to a lobe ramifies widely and has numerous anastomosing branches; the pulmonary artery to a lobe branches into end-arteries which have only capillary communications. Normally blood from either system does not enter the other.

3. Under abnormal conditions certain invariable changes in the two systems occur as demonstrated in the following studies:

A. Pneumonia: (a) The bronchial artery to a lobe involved in pneumonic changes becomes definitely dilated. (b) The pulmonary artery fails to fill completely in the precapillary area due to thrombosis or occlusion by pressure from surrounding edema. (c) The lymphatics to the affected lobe are dilated and filled with blood-tinged fluid.

B. Atelectasis: The bronchial artery and pulmonary artery are normal in the presence of sterile atelectasis. In the presence of pneumonia in the atelectatic lobe, dilatation of the bronchial artery and incomplete filling of the pulmonary artery in its precapillary bed occur.

C. Embolism: Occlusion of a branch of the pulmonary artery to a lobe invariably led to marked dilatation of the bronchial artery to that lobe and the pulmonary artery in the embolic area eventually was fully supplied with blood, usually by way of the bronchial artery, or occasionally by dilatation of capillary anastomoses with the pulmonary artery adjoining the embolic area. Due to this prompt and adequate nutrition of the lung beyond a pulmonary embolus, little evidence of this embolic area was present macroscopically if the embolus was sterile. If the embolus was infected changes in the pulmonary parenchyma invariably occurred ranging from a localized pneumonitis, hemorrhagic infarction, to abscess formation and hemorrhagic consolidation. The bronchial artery was invariably dilated to a marked degree in the presence of an infected embolus and was undoubtedly an important factor in the repair of the diseased tissue.

D. Following ligation of the pulmonary artery to a lobe, slight shrinkage of the lobe occurred in the absence of infection and the lobe showed little macroscopic change. The bronchial artery, however, was invariably markedly dilated and the pulmonary artery beyond the embolus was filled through the dilated bronchial artery.

Clinically, it may be concluded that the bronchial artery serves an important function following small or large pulmonary emboli, by filling both circulatory beds beyond the embolus with blood, and in case of infection providing increased nutriment to the affected lung by virtue of its dilatation.

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DISCUSSION

DR. BUTLER.—The dual circulation in the lungs has considerable physiologic and pathologic significance. Large volumes of deoxygenated blood are poured in through the pulmonary arteries, but only very small amounts of oxygenated blood enter by way of the bronchial arteries. If the air passages are occluded, the affected lung becomes the most poorly oxygenated tissue in the body. The luxuriant growth of anaerobic organisms bears witness to this fact. Dr. Holman, in showing an actual increase in supply of oxygenated blood through the bronchial arteries, together with decrease of blood flow through the pulmonary arteries, has suggested a p[illegible]iologic explanation for the benefit which follows compression therapy.

DR. STEPHENS.—I would like to ask Dr. Holman how he explains the increased circulation, which occurs in the lung after a collapse has already been produced. There was an increase in the blood flow in the bronchial artery in those two cases that you mentioned, which increase prevented the breaking down of that lobe.

DR. BRUNN.—I should also like to ask Dr. Holman a question, particularly with regard to the development of the atelectasis in his cases. I was interested in knowing the anesthesia used, and whether the animal was quiet under the anesthesia.

DR. HOLMAN.—Infection was the invariable cause of dilatation of the bronchial artery. Sterile atelectasis unaccompanied by subsequent infection was not followed by dilatation of the bronchial artery. The sea sponges were introduced under ether anesthesia, with the animals perfectly quiet.

INTERNAL DRAINAGE OF LUNG ABSCESS BY EXTRAPLEURAL COMPRESSION*

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IT IS now established beyond peradventure that in all of us the soiling of the intrabronchial system with bacteria conveyed by air or fluid is an almost daily occurrence. While ciliary action within the bronchi and peristaltic motion of the bronchi contribute much to elimination, yet we know that the widening and contraction of the bronchi with the increase and decrease of intrathoracic pressure and the tremendous power from behind of the air forcibly expelled during an efficient cough is of the utmost importance in maintaining the cleanliness of the bronchopulmonary system. The cough is the body's most efficient means of expelling unwanted material from this tract. Any measure, therefore, that interferes with intrathoracic compression and so with coughing seriously handicaps pulmonary and bronchial drainage. Opening widely the lung for abscess drainage establishes direct and free air communication between the open chest wall and the bronchi and trachea destroying in considerable measure this intrathoracic compression and coughing propulsive force.

A wide zone of pus-filled lobules surrounding a lung abscess may or may not lead to necrosis. With unimpaired coughing efficiency these lobules may be emptied by bronchial drainage before actual death of the local lung tissue occurs. Further extension is then improbable. If the lung abscess is opened externally, however, while the surrounding pulmonitis is extensive the surrounding lobules cannot be emptied by intrathoracic propulsive force, owing to the great reduction in intrathoracic compression incident upon the opening of the bronchial system through the chest wall, necrosis of the lobular walls choked with pus develops, the inflammatory process advances into the surrounding tissues and thus necrosis extends until from toxemia, hemorrhage or intracranial abscess death supervenes.

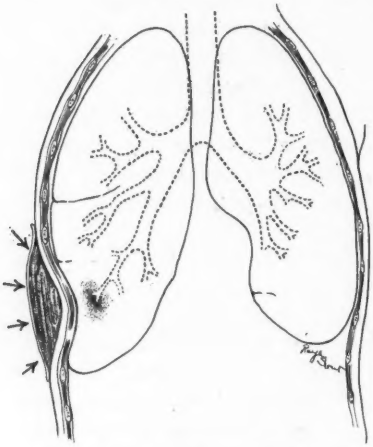
During the acute stage of the first six to eight weeks the drainage of lung abscesses through the chest wall has been uniformly disastrous largely because of the resulting reduction or loss of intrathoracic compression on the involved side. Surgery is contraindicated during this acute stage. After the lobules surrounding a chronic lung abscess have been emptied by intrathoracic compression and propulsion incident upon efficient coughing the results following external drainage have been uniformly good. It is chiefly among those cases where the pneu-

*Read at the Fourteenth Annual Meeting of the American Association for Thoracic Surgery at San Francisco, July 1, 2, 3, 1931.

monitis surrounding the lung abscess is still extensive or continues to extend that we find our great percentage of operative deaths, and it is particularly for this type that the author offers the following procedure:

Its principal is based on continuous extrapleural pulmonary compression against the involved area (Fig. 1) in a direct line towards the draining bronchi to aid collapse and drainage of the lung tissue surrounding the area of liquefaction and necrosis without decreasing to any appreciable extent the intrathoracic coughing efficiency. The technic is as follows:

The location of the abscess with its surrounding pneumonitis is determined by x-ray, the direction of the lobar bronchus draining the suppurative area is determined by bronchoscopy. A site in a direct



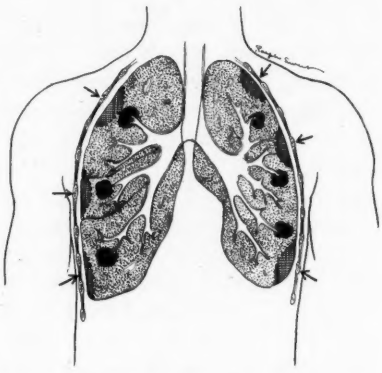


Fig. 1.—Gauze compression against both pleural layers with lateral compression against the necrotic lung area. Note rib periosteum compressed inward.

Fig. 2.—On right side correct method of compression shown in direct line with draining bronchi. On left side incorrect compression tending to narrow the draining bronchi.

line with the bronchus draining the involved area is located at the point where the abscess is nearest the lung surface (Fig. 2). Under local anesthesia short portions of three or even four ribs are excised together with their intercostal bundles (Fig. 3), leaving for the floor of the wound the parietal pleura clear of muscle, vessels, and nerves except for the periosteums of the removed ribs which are left in situ (Fig. 4). The parietal pleura is not freed beyond the limits of the wound, since too great collapse may interfere with bronchial drainage by the falling together of the smaller draining bronchi already softened by inflammation. The wound is packed very tightly with continuous five-inch dry gauze (Fig. 5), and closed without drainage (Fig. 6). The area deprived of rib sections is strapped very tightly with adhesive in order to restore as nearly as possible a solid chest

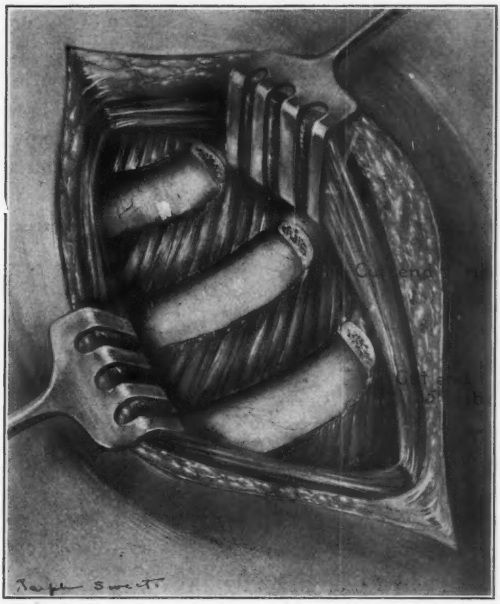


Fig. 3.—Showing local rib resection with periosteum left in situ.

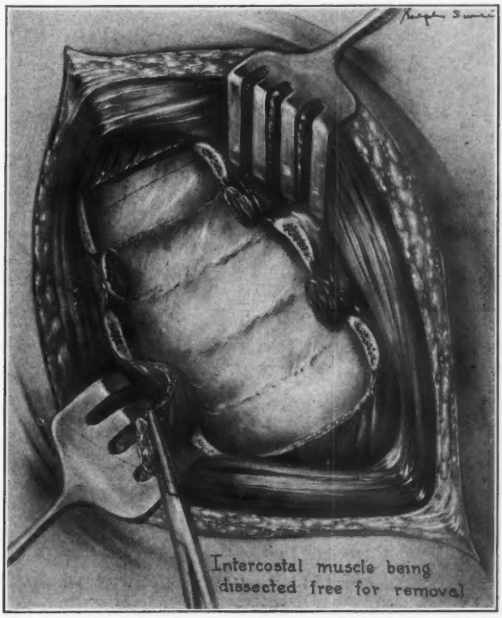


Fig. 4.—Intercostal bundles with nerves and vessels removed. Rib periosteum undisturbed.

wall for counterpressure in coughing. The wound is not opened for fourteen to eighteen days unless infection demands it. The wound is then opened widely and the packing is removed. It is at once repacked with gauze saturated in some antiseptic solution and left wide open. The packing is changed every two days until rib regeneration has firmly occurred with the ribs in the compressed position (Fig. 7). If the collapse obtained by this procedure is insufficient, at a second stage another adjoining area is deprived of its ribs to obtain further col-

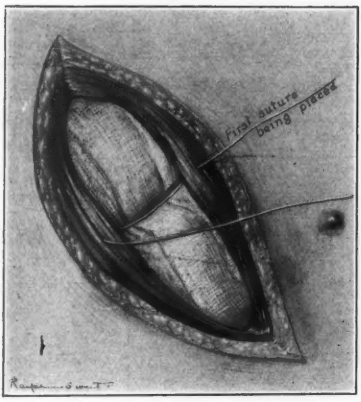


Fig. 5

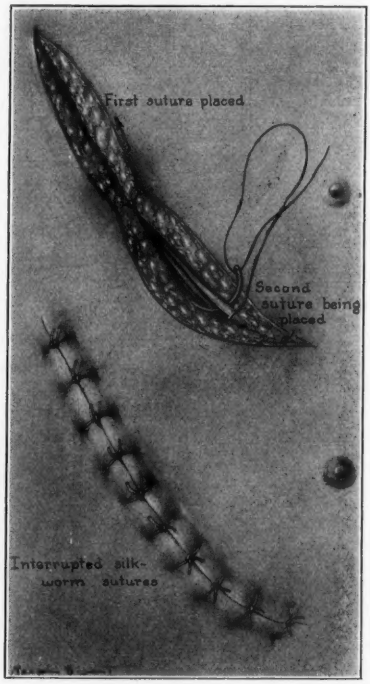


Fig. 6

lapse, and still another stage if deemed advisable. Gauze compression, however, is limited to its original site. After the primary compression operation, further procedures are in the nature of well-circumscribed thoracoplasties, and their number depends upon the area required for collapse to produce continuous improvement to a cure. In one case for multiple abscesses involving most of the lower lobe, I eventually completed a total posterior thoracoplasty, never excising more than three rib sections at a time, however. It is very important that the lung collapse be a gradual one in order to reduce as much as possible the danger of occluding the bronchi draining the suppurative area. While improvement continues following compression or secondary local

thoracoplasty, further stages are postponed until additional chest-wall collapse seems indicated. The clinical course of the patient is a much safer guide than the x-ray films for deciding when and if further surgery is indicated.

There are three situations during lung necrosis in which extrapleural compression therapy has produced dramatic results:

1. Clinically a patient is suffering from a subacute or chronic, low-grade pulmonary infection that incapacitates him. The x-ray shows

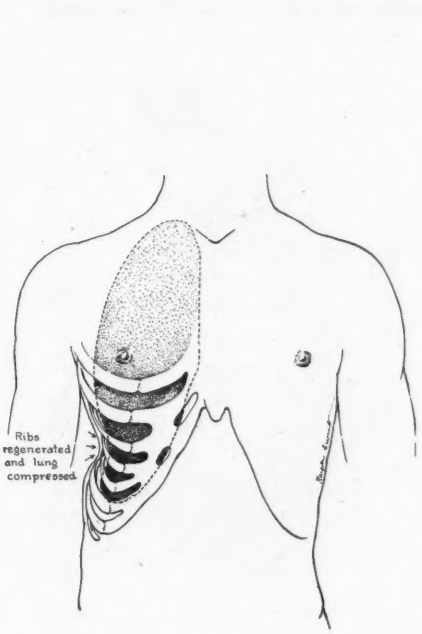


Fig. 7

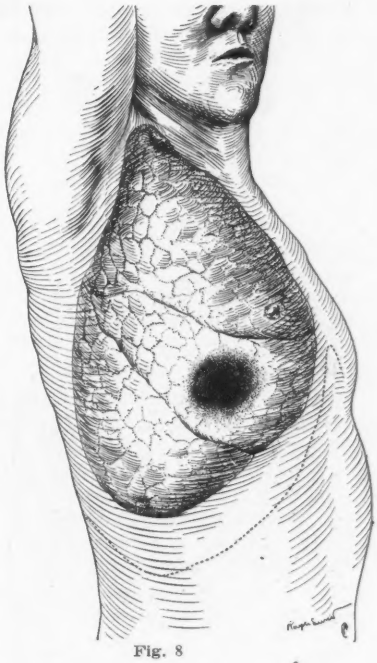


Fig. 8

Fig. 8.—Area of pulmonary necrosis showing neither tendency to heal spontaneously nor to discharge through bronchus.

a well-circumscribed, roughly circular area within the lung without fluid level. The process is often embolic in origin and usually the organisms found are the staphlococcus and pneumococcus (Fig. 8).

P. C., priest, age thirty-two. Tonsils removed under ether anesthesia, October, 1928. Two weeks later bronchopulmonary flare-up, diagnosed pneumonia. Was much better in ten days, though never well since, never a vomica. Temperature ranged from 99-101° daily. Raised small amount of mucopurulent material. Examination repeatedly negative for tubercle bacilli. Seen by me September, 1930. Had lost thirty pounds in weight, was in bed most of day. X-ray showed fairly circumscribed, roughly circular area in center of lung fields on right side. Except for tiny processes extending into lung fields, surrounding lung was apparently clear. With bronchoscope marked irritation of primary middle lobe bronchus was seen, no pus.

On October 3, 1930, portions of the second, third and fourth ribs were excised from midaxillary line forward nearly to costal cartilages. Intercostal bundles removed. Marked compression was exerted with gauze in a line roughly parallel with middle lobe bronchus inwards against abscess region. Wound closed tightly over gauze. Forty-four hours later patient had vomica of approximately one ounce of foul-smelling sputum, containing staphlococcus and pneumococcus, the staphlococcus being apparently the prevailing organism. Wound opened and gauze removed on twelfth day and repacked tightly wide open. Pressure packing continued, changing dressing every other day for eight weeks until ribs were firmly regenerated in their new positions. Sputum slowly but steadily decreased until after eight weeks it disappeared entirely. He is now apparently well, has gained forty-two pounds and has resumed all his old activities.

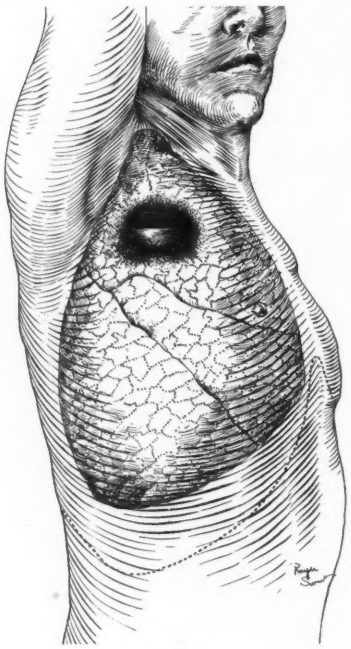


Fig. 9

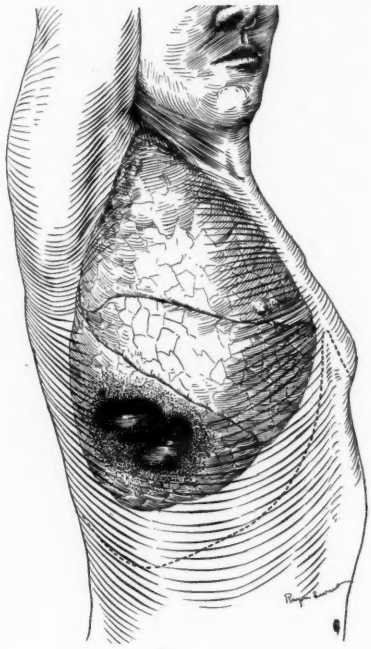


Fig. 10

Fig. 9.—Deeply lying lung abscess with fluid level and wide zone of lobules choked with pus.
Fig. 10.—Gradually extending pulmonitis with multiple areas of necrosis and liquefaction.

2. A vomica of foul pus occurs after a stormy course, a fluid level is evident in x-ray films but surrounding pneumonitis is extensive and shows no tendency to clear up. Frequent stormy exacerbations with a gradual increase in size of abscess and in extent of surrounding pneumonitis (Fig. 9).

F. S., farmer, age 66. Several teeth extracted under gas. Ten days later pulmonary symptoms, diagnosed pneumonia in April, 1929. Was very ill for six weeks. Then had vomica raising a large amount of foul pus. Since then raised

approximately sixteen ounces foul sputum daily. He had several serious pulmonary exacerbations, after each of which sputum has been larger in amount. Process limited to right upper lobe. X-ray showed an abscess in right upper lobe with fluid level about 7 cm. long, apparently extensive infiltration in rest of upper lobe.

In December the second, third and fourth ribs were excised from midaxillary to midclavicular line. Packed in manner described above with pressure as nearly as possible parallel to draining main upper lobe bronchus. Sputum reduced about 50 per cent. General condition much improved. Three weeks later remaining portions of the second, third and fourth ribs were excised anteriorly. Wound closed without compression. Sputum again reduced about one-half, so now, following this operation, raising two ounces daily. Two weeks following second operation first rib divided posteriorly and remaining portions of the second, third and fourth ribs excised. Sputum dropped almost immediately to less than one-half ounce daily. Gradually lessened from this until within three months he was raising practically none. Packing continued every other day for nearly three months. Rib regeneration was slight at end of this time; packing therefore continued. After another six weeks the opening for the packing had closed down to a comparatively small sinus. Rib regeneration had not been marked. When pressure packing was relaxed patient, after four or five days, would begin to raise a little purulent material, one or two drams a day. It is evident that solid rib regeneration is not going to occur, therefore packing is to be continued until the abscess walls are entirely replaced by scar tissue. Patient is back to normal weight and his general condition is splendid.

3. A vomica occurs, sputum is abundant and surrounding pneumonitis is very extensive (Fig. 10). While waiting for the neighboring pulmonary tissue to clear up, other areas of liquefaction develop. The x-rays show several fluid levels, the patient is steadily growing worse.

J. M., tradesman, age forty-seven. Tonsils removed under ether anesthesia. Seven days later acute pulmonary condition developed, diagnosed pneumonia. Was seriously ill for three weeks when he had a vomica of about four ounces of foul pus. X-ray showed fluid level in right lower lobe. June 1, 1930, phrenic interruption without improvement. Patient refused external drainage. Seen September 8. At that time patient was very ill, temperature 103° daily, raising more than 16 ounces of foul pus. X-ray showed five separate fluid levels and pneumonitis limited to but apparently involving most of right lower lobe. Bronchoscopy showed pus coming from all secondary bronchial divisions of this lobe.

Sixth, seventh, eighth and ninth ribs were excised from anterior to posterior axillary line, wound pack. Sputum decreased about 30 per cent. Temperature following this was definitely lower, general condition much improved. Improvement continued. Six weeks later the sixth, seventh, eighth and ninth ribs were excised from anterior axillary line nearly to costal cartilages. Little change seen in patient following this. Two weeks later the sixth, seventh, eighth, ninth and tenth ribs were excised from spinous processes to posterior axillary line. Some improvement following this. Sputum reduced 20 per cent. General improvement of patient from beginning of operative procedures very marked.

Two weeks following this last operation the fifth and fourth ribs were excised posteriorly from spinous processes for a distance of 12 cm. Little change in patient's condition following this.

Ten days later the third, second and first ribs were divided. Following this last procedure improvement was marked. Sputum dropped 50 per cent to about 4 ounces a day. Temperature for first time approximated normal, appetite ravenous.

Since that time patient steadily improved, except for three flare-ups. Now he has gained thirty-two pounds. Is raising less than one ounce a day, is up and about. Has not as yet returned to work.

No lipiodol has been given but feel that probably a considerable degree of bronchiectasis is largely responsible for the present expectoration.

But fourteen cases have been treated by extrapleural compression. All had had prolonged and good medical care. Thirteen of these fourteen are recovering. Nine are sputum free. One died. This patient was among the earlier ones. He should have been drained externally, since there was little surrounding pneumonitis. Enthusiasm for compression therapy, however, delayed external drainage too long. After opening the abscess he developed multiple areas of necrosis in the contralateral lung.

While improvement under rest and postural drainage and bronchoscopy is continuing, surgery should certainly not be considered. When improvement ceases and the abscess is well circumscribed with little circumferential pneumonitis, external drainage is the procedure of choice even though the drainage opening requires usually many months to heal. But when after two or three months of serious illness the lobules widely surrounding an area of pulmonary necrosis are still choked with pus that will probably result in further necrosis if intrathoracic compression and the coughing act are interfered with by external drainage, it would seem that continuous extrapleural compression in a direct line with the bronchi draining the involved area is the most rational and safe procedure, thus helping rather than hindering Nature in reestablishing the protective function she has found most efficient in obtaining and maintaining a clear bronchopulmonary airway.

A series of fourteen cases is not sufficiently large, of course, to make conclusions drawn therefrom of definite value. This is but a preliminary report, but in view of the good results obtained it seems worthwhile and is submitted in the hope that the principle will be given a thorough trial according to each surgeon's idea as to how it may be most successfully applied.

DISCUSSION OF PAPERS BY DOLLEY AND ELOESSER

DR. LOCKWOOD.—I should like to suggest a method that I have used in cases of cavitation in the apex of the lung. In one case I used a lipoma which I was fortunate enough to get from another patient, and I left it in. After resecting two ribs, the second and third, the apex was freed completely. I then planted the mass of fat and left it in. That case was satisfactory.

The idea of using paraffin and wax has never appealed to me very much. It always seemed to me that a slough would almost certainly result, and it would have to be taken out later.

Gauze has never seemed to me a very practical or satisfactory material because infection almost certainly results and it has to be removed. I conceived the idea, in one of my cases in which I wanted very much to get compression, of using vaseline gauze. I used about five yards of vaseline gauze, such as we use for

dressing wounds, and completely packed the area left after separating the apex with its cavity.

This vaseline gauze can be moulded beautifully to fit the cavity which you have freed. In one of these cases I left it in. In the other case I left it in for about three weeks and then gradually removed it. In both of these cases the sputum has been greatly diminished, and apparently the results have been good. I would like to suggest it as a means of compressing a cavity where other suitable material is not available.

DR. TOREK.—I should like to ask Dr. Eloesser a question, and that is based on the uncertainty as to what we should introduce for the purpose of producing extrapleural compression. It seems to me there is objection to almost everything that has been tried. Now, I wonder whether, with a considerable loosening of the outside pleura, it would not do to put nothing in at all but to leave an open air space which could be renewed in the same manner that intrapleural pneumothorax is filled. I don't know whether this will work out.

If in the presence of large apical cavities we would want to release the lung over a considerable area, I should think that this could be accomplished by a procedure similar to the one I have recommended for extrapleural esophagectomy: long incision through the sixth or seventh intercostal space down to but not through the pleura, separation of the parietal pleura from the thoracic wall in a direction upward to and around the apex of the lung. The long incision permits the introduction of the whole hand after the separation of the pleura has proceeded for some distance. The resulting collapse would last at least long enough for the wound to heal and then could be maintained by refills of the pneumothorax.

I would like to hear how that impresses the gentlemen who have tried the other method.

DR. ELOESSER.—The external drainage of abscesses has been so satisfactory that I should dislike to forego it. It seems to me that all other methods of approaching abscesses by extrapleural compression and similar methods are more or less makeshift. If one waits for the acute stage of progressive pneumonitis to subside so that there is a localized abscess cavity, drainage to the outside is so little provocative of complications and so satisfactory that there is really no substitute for it.

I intended this subcostal operation to act as a first stage. Rather than do the rib resection first, I introduce the pack below the ribs and do the rib resection afterwards. Make the incision, separate the periosteum, put in the pack—vaseline gauze as Dr. Lockwood proposes, or paraffin, or whatever substance you like to use—leave it there until the pleura is adherent, then, after two weeks or so, resect the ribs and take the pack out. It takes but a very few minutes to resect the ribs at the second stage. In the meantime you preserve the integrity of the chest cage. This is where the procedure differs radically from Dr. Dolley's.

As for Dr. Torek's remarks: extrapleural pneumothorax has been tried, but the air does not remain. It seems to be coughed out into the subcutaneous tissues and is rapidly absorbed.

I differ from Dr. Holman's idea, as I said, in that I think the drainage of abscesses should be to the outside, provided they reach the chronic stage.

Bronchoscopic treatment of abscesses, unless of those caused by a foreign body, is usually unsatisfactory. The bronchoscope is not entirely innocuous in the treatment of abscesses.

DR. DOLLEY.—Following Dr. Eloesser's discussion I feel it wise to emphasize again that extrapleural compression for internal drainage of lung abscess is distinctly not indicated in those cases of lung abscess where the lobules surrounding an

area of necrosis have in a large measure become clear. There external drainage is definitely the procedure of choice. Extrapleural compression is indicated, however, in those cases in which the surrounding lobules are choked with pus and where there is little or no apparent tendency for this suppurative process surrounding the area of necrosis to clear up. External drainage in such cases is as a rule disastrous whereas external compression without disturbance of intrathoracic coughing efficiency has in the comparatively small series quoted in my paper given almost uniformly good results without any evident increased danger to the patient from the operative procedures themselves.

DR. HUDSON.—I have been very much interested in Dr. Dolley's remarks in regard to the treatment of pulmonary abscesses. He has emphasized some of the points that we ourselves have tried repeatedly to emphasize. One of these points, is the fact that the important thing we are after in the treatment of any suppurative lesion in the lung, is drainage, and that it is not so important as to how the drainage is obtained as it is important that we obtain the drainage. It is very important, however, that the drainage obtained be continuous and that it is sufficient to remove any accumulating secretions that may be present or may form.

Dr. Dolley mentioned the importance of conservative measures in the treatment of pulmonary suppurations and, as he has told us, conservative measures should be tried even before resorting to the methods that he has spoken of today. I am of the impression that at least in certain communities the importance of bronchoscopic drainage along with postural drainage is not fully realized, and I am of the further opinion that where it is used the use is not energetic enough.

We have found that the use of the bronchoscope in the treatment of lung abscess will in a majority of instances entirely satisfy our needs if it is used at very frequent intervals with the idea of maintaining a drainage canal that is always open and free of obstructions.

Dr. Holmes showed us this morning a case of an abscess of considerable size, which reminded me of a case we have recently had in which there was an abscess of enormous size. We passed the bronchoscope every other day at first, using postural drainage between times. Later the bronchoscope was passed once a week. A completely satisfactory result was had within a period of about three months.

Now, Dr. Jackson and his group have pointed out the fact that obstruction is produced in the bronchi through the development of granulation tissue and through the thickening of the mucosa by edema and inflammation, and that it is only by the repeated passage of the bronchoscope that the drainage canal is kept open. Our experiences have been such that we are very enthusiastic in regard to the use of the bronchoscope in the treatment of pulmonary suppurative lesions. I bring this subject up again at this time because Dr. Dolley, although he mentioned it, did not emphasize its importance as strongly as I feel that it should be emphasized. This lack of emphasis we felt was due to the fact that he was discussing a type of abscess which has reached a stage in its development that makes it less suitable for bronchoscopic treatment than are the abscesses that are seen at an earlier stage in their development.

BRONCHIAL STENOSIS*

(Continued from p. 295)

L. ELOESSER, M.D., SAN FRANCISCO, CALIF.

III.

PHYSICAL EXAMINATION

ON *physical examination* of patients with *stenosis of the trachea or at the bifurcation,* dyspnea and often a stridor are evident. They tend to sit up with the chin stretched forward. In the compensated stage respiration is slow and labored; expansion of the chest may be increased, although evidently at the cost of much muscular effort. The effort and resistance to respiration are shown by deep inspiratory retraction of the intercostal spaces, often very noticeable.

Gerhardt draws attention to a sign known by his name; viz., in tracheal stenosis the larynx moves but slightly in comparison to the violent laryngeal excursions when obstruction lies at the level of the glottis. Laryngeal excursion of less than one cm. is said by him to be pathognomonic of lower stenosis.

This picture changes during the suffocative stage when the respiratory rate is increased, intercostal retraction not so noticeable and breathing much shallower. Chest expansion is reduced in spite of the effort with which it is accomplished.

The chest is of normal resonance or hyper-resonant; breath sounds are diminished and accompanied by dry squeaky ronchi and râles. They may, however, be tubular or amphoric and fairly loud.

These signs may be complicated or obscured by older underlying or concomitant intrathoracic processes; old tubercular cavities or pleurisy, effusions caused by malignant pleural metastases, the dullness of a large aneurysm or mediastinal tumors and abscesses.

The peripheral veins are often congested and the extremities bluish. The face is inclined to be cyanotic and there may be some fullness of the subcutaneous veins of the chest. The right heart may be enlarged.

The liver may be enlarged. Edema of the extremities and fluid in the chest or abdomen are rare in patients who are not toxic.

Minor localizing signs are of value. Pupillary differences may point to sympathetic irritation and hoarseness to laryngeal palsy. Signs of syphilis, penile scars and adenopathies should be looked for.

There may be constitutional signs of tuberculosis or tubercle bacilli in the sputum.

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*From the Department of Surgery, Medical School of Stanford University, San Francisco Hospital.

Read before the Thirteenth Annual Meeting of the American Association for Thoracic Surgery, Philadelphia, May 19, 1930.

Tumors may produce palpable metastases in the ribs or sternum and not infrequently axillary or supraclavicular glandular enlargements.

The back should be examined for deformities that might cause an abscess with perforation into the mediastinum, Pott's disease or other forms of chronic vertebral osteomyelitis.

Direct evidence is given by the laryngobronchoscope, which reveals the site, extent, and often the nature of the stricture. Tracheobronchoscopy is the sovereign diagnostic method for upper stenoses; it should not be omitted. A high stenosis may be visible in the ordinary laryngoscopic mirror.

The x-ray is of indirect value. It usually shows only concomitant processes; calcified or enlarged hilar glands, tumors, aneurysms, mediastinal abscesses. It is rarely possible to outline the trachea and bronchial tree sufficiently clearly to allow of a positive diagnosis of constriction; occasionally, however, especially when imbedded in dense glandular masses the trachea and major bronchial lumina stand out with such clearness that indentations with adjacent shadows are visible in their course, justifying suspicion at least of an extrabronchial mass and a pressure defect. These defects are made still clearer by allowing a little lipiodol to trickle along the tracheal wall and viewing it both under the fluoroscope and in the film. It is often best seen on the screen. The lung fields themselves are not radiographically characteristic, although retarded or impaired excursion of the chest wall may be seen under the fluoroscope as it is at the bedside.

An opaque catheter introduced through a bronchoscope or an opaque medium through the instrument directly onto the stricture (after the method of Jackson) may help very considerably in the recognition of topographical relations between a presumably stenosing shadow and its pressure defect. Stereoscopic films should be taken both anteroposteriorly and from both sides.

Complete stenosis of a major bronchus offers the very striking signs of massive collapse. The affected side of the chest is shrunken, the ribs lie close together, there is not infrequently, especially in children, a scoliosis with its convexity toward the affected side. The intercostal spaces may be retracted if the ribs are not pushed too closely together to allow of retraction. The side stands still. The thoracic viscera are pulled toward it; the heart is deviated; the trachea enters the chest toward the bad side. The affected diaphragm lies high and stands still, or it may rise on inspiration (paradoxical movement) and move down jerkily on expiration. In recent stenosis a movable mediastinum may deviate toward the affected side on inspiration and swing back to the midline on expiration. This deviation, known as Jacobson[62]-Holzknecht's[63] phenomenon, was most strikingly observed by Pfeiffer[64] who occluded the right major bronchus with a sponge introduced through a bronchoscope. Immediately he saw the medias-

tinum deviate to the right over a distance of 6 cm. The deviation lasted as long as the patient held his breath in inspiration. Deviation no longer occurred when the sponge was pushed down further, occluding only the lower lobe bronchus but leaving the main one free.

The obstructed chest is dull or flat. Breath sounds are absent or much diminished; when heard at all they are tubular.

The signs then are those of effusion except for retraction of the chest and its contents.

These are the characteristic signs. Again there are minor ones. The intercostal spaces may be tender from an underlying pleurisy; there may be perforating sinuses from distant foci of suppuration; there may be distant localizing signs, pupillary differences, hoarseness, etc., as noted above. Aneurysms, intrathoracic tumors, cysts and mediastinal swellings may document themselves in various ways. Dyspnea is ordinarily not excessive and not characteristic. It is increased by exertion but is not to be distinguished from that caused by pleural effusions and similar processes reducing the vital capacity.

Neither is the sputum characteristic. In intermittent stenosis it may undergo sudden variations as mentioned above; it may be bloody at times or purulent or foul; thick and nummular or thin.

Bronchoscopy usually reveals the obstruction, sometimes its nature. The stricture itself is seen and may be entered with a bougie; sometimes a bulging of the bronchial wall appears, giving evidence of pressure from without, sometimes a tumor mass or an ulcerating granuloma, bits of which may be removed for microscopic examination. A history of hemorrhage will make the bronchoscopist doubly cautious when introducing his instrument and hesitant about removing bits of tissue.

X-RAY

X-ray examination is characteristic, complementing physical examination and the bronchoscope. One side of the chest is gray and airless; its diaphragm is raised and moves but little if at all or moves paradoxically; the ribs of the affected side lie closer together; the thoracic viscera are drawn toward the affected side; the trachea is curved or kinked toward this side; the heart and great vessels pulled toward it. It has seemed to me before the fluoroscope that mediastinal deviation decreases on coughing, i.e., that an increase of intrathoracic pressure tends to let the mediastinum move back toward the midline. This movement is difficult to make out with precision on account of the greater filling of the great veins after cough. Occasionally the cause itself of stenosis, a tumor, an aneurysm, a large chalky gland, is visible (Case 5); occasionally as in higher stenosis the hilum tissues are dense enough to furnish a good contrast to the air-containing bronchi imbedded in them; such films will at times

reveal the bronchial lumen fairly accurately, and occasionally even justify the surmise of a deviation or constriction.

It is well to make two sets of films; a normal exposure for the good side, a heavy one for the stenotic side. A heavy exposure will often reveal details, chalky masses, cavities, etc., that remain concealed in the shadows of a normal exposure.

Injection of lipiodol (iodized oil) into the air passages gives information exceeded in accuracy only by the bronchoscope. The oil must not be too warm; the thicker it is the better. Its flow should be watched with the fluoroscope; seen in its transit down the trachea it will often show narrowing and deviation that the film may be too late to catch. It should be injected a little at a time, not more than 3-5 c.c. The patient sits before the screen with his tongue out; the back of the tongue and the anterior faucial pillars are swabbed with a 5 to 10 per cent cocaine-adrenalin solution a little of which is allowed to trickle down into the air passages. Lipiodol is injected and its course watched. It flows down to the level of the stenosis and stops there. The occluded bronchus fills to the level of the carina after which additional oil spills over the top of the bifurcation into the sound side. A thin stream of oil often trails slowly through the stenosis down the distally dilated bronchi and puddles at their bottom. Much of this interesting picture is lost if one trusts to films alone. They can scarcely be taken quickly enough to follow progress of the oil in all its details. Films should be exposed sufficiently to bring out the lipiodol through a dense chest; lung detail is of no importance. An ordinary chest exposure is insufficient; details of the injection are lost behind the shadow of an airless lung. Too much lipiodol obscures detail instead of helping to outline the bronchi; about 10 c.c. is usually enough, sometimes 5, rarely as much as 15; the less used and the closer it is put and kept at the site of disease the better.* Operation may furnish decisive evidence to corroborate a suspicion or confirm a previous diagnosis. When an open bronchus is entered, as at operation for bronchiectasis or abscess, the patient immediately coughs and blows out a great quantity of air mixed with blood or pus through the wound. No cough ensues when the periphery of a stenotic bronchus is opened, and no air comes through the wound when the patient blows or coughs. (Cf. Case 17.)

Examination of *intermittant unilateral stenosis* gives rise to the most striking variations; very confusing until one is aware of what to expect. On one day all signs of total obstruction may be present—immobility of one side of the chest, a high diaphragm, mediastinal devia-

*A little trick of technic: when taking films it is well instead of ordering the patient to "Take a deep breath and hold it" to say simply "Don't breathe." Taking a deep breath often provokes a cough which ejects the lipiodol from the bronchi and sprays it about in the alveoli. This dispersion not only blurs the film but spoils the lung for further injection.

tion, flatness, and absent breath sounds, together with distressing nonproductive cough, fever, sweats, and considerable dyspnea. The next day these may be gone; the chest may be movable, the flatness may have given way to resonance, breath sounds may be heard, accompanied usually by coarse bubbling râles, the patient will be coughing or have coughed up large amounts of sputum; fever, dyspnea, and the feeling of tightness will have disappeared. The change is ordinarily not so dramatic or so rapid as that seen in acute massive collapse, which may clear in a matter of hours. In chronic stenosis intermissions are rarely so complete or so rapid. However, stenotic patients followed over a considerable period rarely fail to show an easily recognizable degree of variation and remission even though the chest may never be quite free or completely blocked. The block may occur quite suddenly, within a day, occasioned by a slight superimposed infection or other indeterminable cause; it may last for several days or weeks. Recession takes place more slowly; one can watch the fever drop, the sputum increase, the chest clear, excursions grow wider, flatness give way to dullness and finally perhaps to resonance. Breath sounds come through and the grayness visible in the x-ray gives way more or less rapidly to translucency. There may be months' interval between recurrences; two patients have worked a year in lumber camps and ranches between attacks without serious disability, although "their wind was never quite what it should have been."

CASE 19.—Deforming bronchitis. A Swedish miner and lumber man of twenty-eight was admitted to the San Francisco Hospital on November 22, 1925. On leaving Alaska in 1922 he had begun to complain of a dry unproductive cough, shortness of breath and pain in the left chest. He had spent all but four months of the years 1922-25 in hospital. He had two attacks of fever, attributed to "pneumonia," in which his cough was productive; otherwise he had brought up nothing.

Prior to admission to the San Francisco Hospital he had worked for a few months, but had quit ten days before on account of chills, fever, and uncontrollable paroxysms of cough with very little expectoration.

On examination the left side of the chest was much contracted and dull, especially posteriorly and in the axilla. Over the left apex the breath sounds were cavernous. There were râles over both lungs. The heart was retracted far to the left, the apex being felt in the fourth intercostal space just behind the anterior axillary line. Repeated puncture of the chest brought forth no fluid. X-rays of December 17, 1925 were reported as showing a thickened left pleura, especially at the left base, with wide and dense hilar shadows and numerous calcified hilum glands. Both apices and the area of the first right intercostal space were a little mottled.

Lipiodol injected into the trachea puddled in the left main bronchus about 2 cm. beyond the bifurcation and, as more was injected, filled the bronchus to the bifurcation and spilled over into the right side. Films showed that a little lipiodol had found its way through the stenosis and entered the left lower lobe. Bronchoscopy by Dr. Graham revealed a normal right bronchial tree and a blocking of the left major bronchus by a firm fibrous mass which did not bleed when touched. Exploratory thoracotomy was performed April 1, 1926, after preliminary pneumothorax. The right hilum was surrounded by enlarged lymph nodes. Manipulation

of these nodes provoked an uncontrollable cough which made further exploration impossible. The chest was closed. The patient was considerably shocked by the operation, but recovered and was relieved of his dyspnea. Bronchoscopy a month later showed that the obstruction to the left bronchus had practically disappeared. The bronchoscope passed through the area which had formerly been stenotic, so that the smaller branches could be explored. The only abnormality noted was some congestion of the mucosa.

The man brought up from 30 to 55 c.c. of purulent sputum daily. Seventeen examinations of this expectoration failed to reveal tubercle bacilli. The Wassermann reaction twice repeated was doubtfully positive.

He continued to improve and got about the ward with little dyspnea. On April 14 the left lung field was still gray, but the trachea and the heart occupied almost normal positions; on May 26 and June 24 the lung was much better aerated.

He was discharged May 27 and went to work as a waiter in a lumber camp. He reentered hospital November 26, 1926. He had been somewhat out of breath,

Fig. 35.—Case 19. Lipiodol injection of trachea, stenosis of left major bronchus. A spot of lipiodol seen filling origin of left major bronchus; the remainder has spilled over into right base.

had coughed a little since his discharge, but had had no severe paroxysms and no fever up to five days ago when he was taken with chills and a temperature to 104° F. This lasted for two hours when he coughed up a large amount of green sputum and the fever dropped to 101° F. It had risen to 103° F. since. The left chest was again dull, the breath sounds diminished or absent. Lipiodol injected into the bronchi again stopped at the site of stenosis and bronchoscopy revealed a stenotic left bronchus with a very small opening from which pus exuded. The temperature dropped, but on November 30 rose again; the coughing fits and dyspnea grew more severe, the pulse was rapid and over the left upper chest loud tubular breathing with bubbling râles could be heard. The man died on December 3, 1926.

At autopsy the next day the heart was found to lie almost entirely on the left side of the body; the right lung was emphysematous and projected a little beyond the midline. The left pleura was obliterated by strong adhesions. There was a narrow stenosis of the left main bronchus 2-3 mm. in diameter. At this point the bronchial wall was 4 mm. thick as against 2 mm. in the unaffected portion. The mucosa was smooth, gray and much congested; at the narrowest portion it was somewhat scarred. There was no induration of the tissues around the stenosis, but right close to it lay some soft lymph nodes, the largest 2½ cm. in diameter, some grayish red, some light gray with light spots.

The left pleura was very thick, the left lung very small. The apex was free of scars. The bronchi distal to the stenosis were dilated and filled with a dirty brown fluid. The bronchi of the upper lobe were also more or less dilated; the lung was dark red and almost solid. The right lung was very large, emphysematous; the posterior two-thirds of the lower lobe and the posterior half of the upper lobe were consolidated. The middle lobe was free; the bronchi were not dilated.

The heart was of normal size; the right half was dilated. Sections of the peribronchial lymph nodes revealed marked congestion and hyperplasia and slight capsular thickening. There was no evidence of tuberculosis.

Fig. 36 Fig. 37

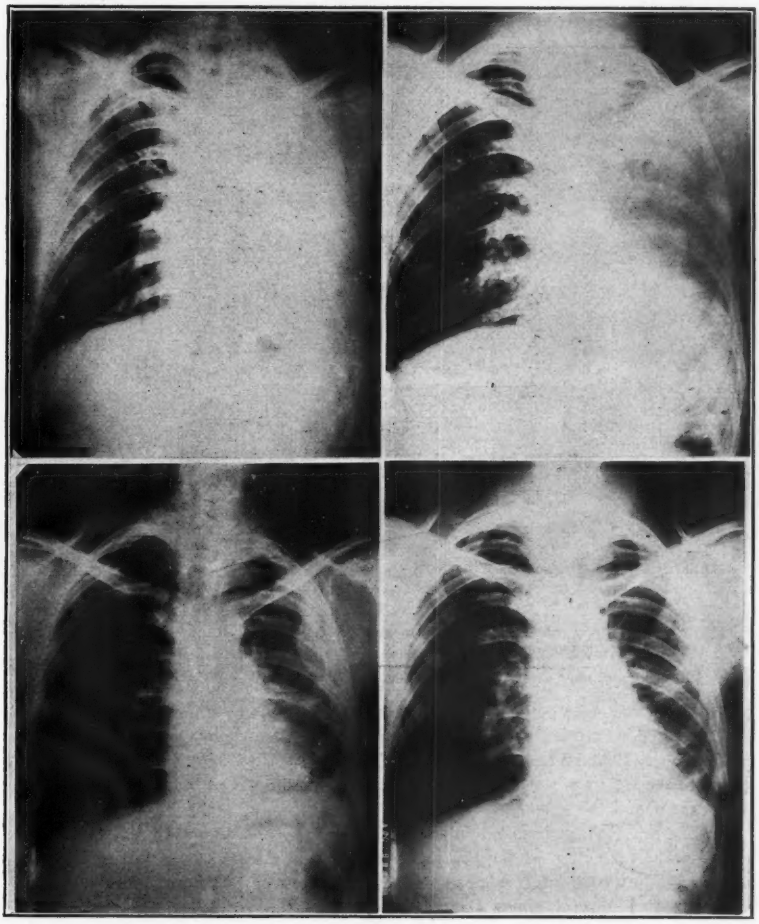


Fig. 38 Fig. 39

Fig. 36.—Case 19. Feb. 18, 1926: Gray left chest; marked tracheal deviation.
Fig. 37.—May 7, 1926: Same; beginning aeration.
Fig. 38.—May 25, 1926: Same; aeration progressive.
Fig. 39.—June 26, 1926: Same; aeration practically complete.

The diagnosis was made of stenosis of the left major bronchus, bronchiectasis and terminal pneumonia.

CASE 20.—Syphilitic bronchial stenosis. A Danish seaman of fifty-one first entered the San Francisco Tuberculosis Hospital on June 3, 1924, on account of cough, expectoration, and pain in the right chest. He had had a chancre twenty-one years ago. He brought up 250 c.c. of purulent sputum per day in which no tubercle bacilli could be found. He had no fever. There were many râles over the right middle lobe and much wheezing all over the chest. The Wassermann reaction was positive. The x-ray was interpreted as showing cavitation of the right middle lobe and tuberculosis of the left lung with cavitation. He was discharged on June 24, 1924; the clinical diagnosis was withheld. He reentered on November 25, 1924, with the same complaints and was given twelve injections of neoarsphenamine. He again

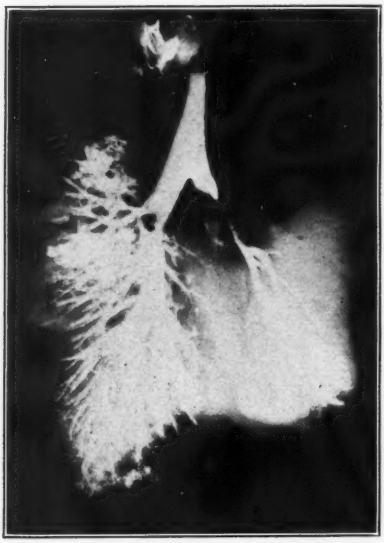


Fig. 40

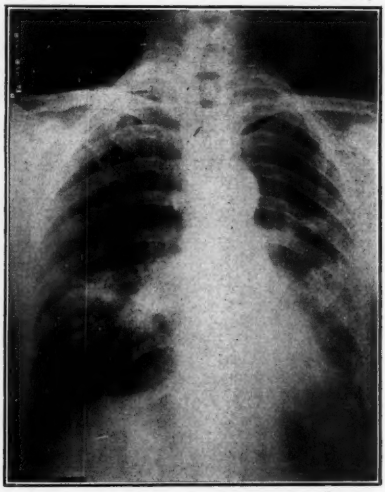


Fig. 41

Fig. 40.—Autopsy specimen; barium gelatin injection; stenosis left major bronchus; peripheral bronchiectasis.
Fig. 41.—Case 20. Syphilitic intermittent stenosis of left bronchus and of right middle lobe bronchus. Left chest contracted; right middle lobe gray.

improved, was discharged and remained fairly well up to June 3, 1928, when he again entered hospital. He had weakness, night sweats, dyspnea and had lost weight. In December, 1928, he had fever to 140° F. The left chest lagged. The trachea was pulled to the left; the left base was dull. There was bronchial breathing between the left scapula and the spine.

X-rays repeated at frequent intervals disclosed a much shrunken left hemithorax with marked retraction of the heart and mediastinal viscera to the left and a grayness at the left base interpreted as due to an atelectatic left lower lobe. Bronchoscopy repeated several times showed scarring at the carina which extended for some distance down both major bronchi, with mucopurulent secretion coming from the left side. The mucosa of the left bronchial tree was swollen and red. The lumen of the left major bronchus was distorted, but not closed. Antisyphilitic treatment led to no improvement and the patient is at present an inmate of the Relief Home.

CASE 21.—Syphilitic bronchial stenosis. A railway trainman some forty years of age with a strongly positive Wassermann reaction, was examined at the United States Veterans' Hospital at Palo Alto on February 21, 1930, because of dyspnea, cough and expectoration.

Bronchoscopy: The bronchoscope enters without difficulty. The larynx is normal; the upper trachea is normal. The carina is seen as a blunted, reddened ridge, and the instrument looks into the right major bronchus. The lumen appears normal although not quite rounded. It is a little flattened on the right side. It is impossible to make out a left major bronchial lumen, even when the head is inclined strongly to the right. It may be that a lumen lies under some of the projecting tracheal rings. Numerous attempts are made to push a ureteral catheter forward at a site where the lumen might be supposed to come off. None of these is successful. No scarring or other obstruction of the lumen is visible. The left wall of the trachea down to the blunted carina is formed by a series of projecting tracheal rings, covered by normal, slightly reddened mucosa.

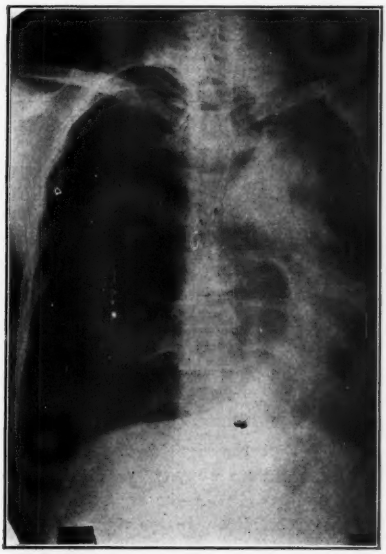


Fig. 42

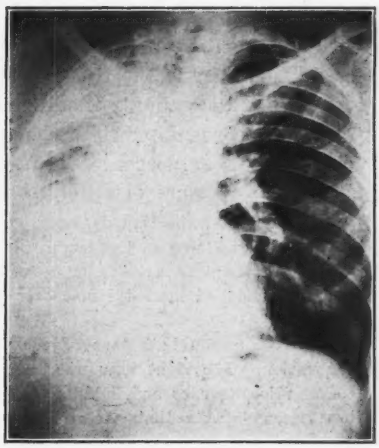


Fig. 43

Fig. 42.—Case 21. Syphilitic stenosis, left major bronchus. Extreme retraction of mediastinum, the right border reaching to left nipple line. Extreme elevation of left diaphragm, which lies five intercostal spaces higher than the right. The heart is correspondingly raised and displaced to the left.
Fig. 43.—Case 22. Marked contraction of the right chest. Airless right lung. Trachea and mediastinum displaced to right.

The x-ray revealed a most exorbitant contraction of the left side of the chest. The mediastinum lay in the left nipple line, the right lung filling half of the left chest; the left diaphragm lay five intercostal spaces higher than the right; the heart was correspondingly raised and displaced to the left. Were it not that in former years the man had been repeatedly examined by various railway surgeons and found sound, the bronchoscope and the x-ray would incline one to diagnose a congenital stenosis of the left bronchial tree rather than a syphilitic one.

There is reason to believe that all three forms of unilateral stenosis, total, partial and intermittent, occur more frequently than is suspected. In the x-ray films of every large tuberculosis hospital are films of totally gray chests without effusion—total stenosis—and even more frequent are films showing an enormous amount of unilateral cavitation with a practically free sound side, suspicious at least of partial stenosis. Some series demonstrate great variations in density and in degree of retraction in films taken at various intervals, variations quite characteristic of intermittent obstruction. When pneumothorax is done the manometric needle reveals an extraordinarily low pressure. Such chests are strongly suspicious of harboring a bronchial stenosis as an immediate cause of a large part of the picture even though the stenosis may not be responsible for all the morbid changes they contain. Some of them have already been noted by phthisiotherapists. This is an important matter. It is not unlikely that more frequent use of the bronchoscope will unearth innumerable such stenoses, and that some of them might be helped by its use. Lipiodol, or any other substance injected in the trachea, when sprayed about in the lung by a cough may disseminate tuberculous material. Such injections are too dangerous to make their use in tuberculosis advisable; but the bronchoscope, if unpleasant is harmless and will clear the question of stenosis even more definitely than injection of opaque substances.

Case 22.—A Dalmatian gardener, miner and cement worker was referred by Dr. Knorp of San Mateo on October 6, 1930. In 1909 he had a pleurisy and was very sick for a year and a half, but afterward gained much weight. In 1918 he had influenza and had been dyspneic ever since. He had coughed for the last fifteen years.

On examination both tops of the chest moved, but the right base lagged on respiration. The right base was flat, breath sounds were absent, but one heard coarse râles, squeaks and bubbling noises over this area. Over the right apex the breath sounds were diminished. The heart lay almost entirely in the right side of the chest and the mediastinum was moved far to the right. The left side was free. The fingers were clubbed and the man was very short of breath.

The shrinkage of the right chest, the deviation of the trachea and the heart and the airlessness of the right lung were visible in the x-ray.

The diagnosis of tubercular stenosis of the right major bronchus was confirmed by bronchoscopy. The left major bronchus was seen to make a straight line with the trachea. Its walls were normal and its mucosa was pale. The carina deviated so that its anterior edge was to the right and its posterior one to the left. It was necessary to push the tube far to the left in order to see down the right major bronchus. One now saw that its mucosa was thickened, red and inflamed, so that the cartilaginous rings were indiscernible. Upon lifting the tube far to the front one began to see a lumen. This was full of a viscid secretion, whereas no secretion came from the left side.

Diagnosis: Bronchial stenosis and deformity of the right bronchial tree, tubercular. The bronchoscopy was immediately followed by an avulsion of the right phrenic nerve.

Stenosis of a single lobe or less may cause distinct retraction of the chest wall over the closed area; especially lower lobe stenoses, which

affect the more yielding portions of the thorax. Not only the intercostal spaces but the whole chest wall over an area the size of the hand may be retracted on inspiration while the rest of the chest expands normally. This sign is pathognomonic; a localized pleurisy may cause standstill but not retraction of the chest wall; a diaphragmatic-pleural attachment filling out the complementary sinus may produce inspiratory retraction of the bottom of the chest, rarely, however, over so characteristically circumscript an area as in bronchial stenosis. This sign is not so pronounced over the upper parts of the chest; it is best seen under the scapula. Its mechanism is clear: the stenotic lobe refusing to expand in inspiration with the rest of the lung holds back the chest wall over it.

The intercostal spaces may be tender from an intercostal neuritis or lymphangeitis transmitted here from an inflamed pleura; when they are so tender the chest stands still both because of the underlying pleurisy and the inflamed chest wall.

The area over the stenotic lobe is dull or flat. It may take careful percussion to discover the flatness, for the lobe may be shriveled and concealed beneath its neighbors. This is especially true of the right middle lobe and more especially of stenotic portions of this lobe lying next to the sternum. A totally obstructed upper lobe makes an easily discernible dullness, when however the top of the lobe contains air and only its lower parts are stenotic the dullness may be hidden under the shoulder girdle or under the breast of a fat woman. The dullness of stenosis does not coincide with the topographical outline of a normal lobe; shrinkage and pull as well as compensatory inflation of neighboring parts of the lung both shift the dullness from the normal position and cover it. Breath sounds are impaired or absent over the dull area; in partial stenosis they may be tubular and fairly loud. Adventitious sounds, pleuritic creaks, squeaks, rubs and crepitations are heard; and with a partially open bronchus, bubbling râles, and coarse and finer crepitations. Operation may clinch the diagnosis if no air comes from the bronchi after they are opened.

The *bronchoscope* is invaluable in diagnosis of lower and middle lobe obstruction. As in more central stenosis it reveals the site, degree and sometimes the extent and nature of constriction. It can demonstrate extrabronchial pressure and intrabronchial tumors, ulceration and scars as well as deviation of the trachea and larger bronchi toward the atelectatic lobe. It is less satisfactory in diagnosis of upper lobe stenosis. The instrument being constructed to look only straight ahead but not around a corner shows the upper bronchial mouth as a slit or crescent, but never as a round hole. If a tumor projects beyond the bronchial mouth into the major bronchus it may be seen and at times seized and the site of its origin determined; if, however, the stenosis lies beyond the origin of the upper lobe bron-

chus then the stenosis itself is invisible and the bronchoscope like the x-ray will allow only of secondary deduction but not of prima facie evidence. It may disclose a little blood or pus trickling from an upper bronchial mouth or deviation of the trachea and major bronchus toward the shrunken lobe. About the mouth of the bronchus a localized area of inflammation may be discernible in which the mucosa is uniformly red and thickened, its normal pink and white striping being lost. Invaluable as the instrument has proved in lower and middle lobe obstruction it has not been decisive in affections of the upper lobe. Indeed, deductions made from information it revealed have at times been misleading. Thus, Case 18 was repeatedly bronchoscoped for an upper lobe stenosis. A mass was seen protruding from the upper bronchial mouth. Bits of tissue removed bronchoscopically were diagnosed granulation tissue by a well-known pathologist. A year and a half later another bit proved to be squamous cell carcinoma, which by this time had spread to the hilum glands. Lobectomy was of no avail.

The *x-ray* shows a characteristic density, corresponding often in position and shape, but not in size to the topography of a lobe. The mediastinal viscera are retracted toward it, the trachea especially is likely to be curved or kinked. The diaphragm may be elevated and stand still, or, in lower stenoses, even rise paradoxically on inspiration, as in the Holzknecht phenomenon of phrenic palsy. Retraction of the chest wall may document itself by a narrowing of the intercostal spaces. The stenosing mass, calcified glands, a tumor shadow, an aneurysm may be visible at times, and from its position and that of the bronchi one may be able to deduce obstructive pressure. Actual narrowing of the lumen cannot be made out with certainty at this level of the bronchial tree.

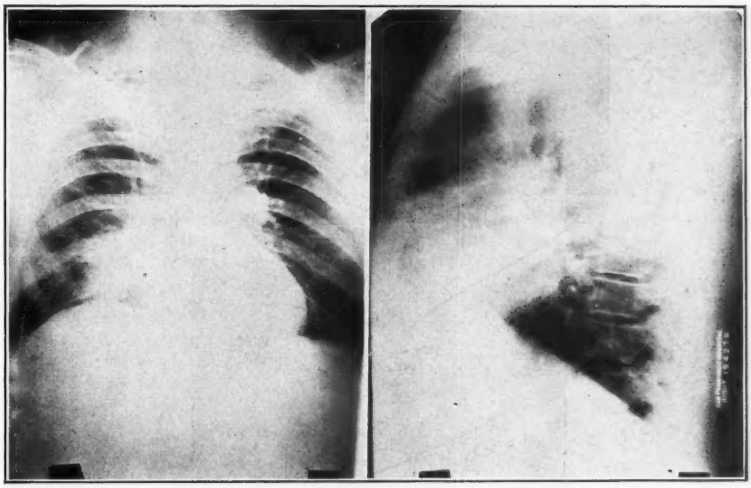
CASE 23.—Stenosis right middle lobe bronchus. An Italian of thirty-six was referred by Dr. McNamara of Bakersfield. He had had a hemorrhoidectomy in October, 1923. Six months later he began to cough and brought up a little blood. He entered hospital for the first time in November, 1924. He breathed somewhat laboriously. There was a retraction of the sternal notch with each inspiration. He was mildly cyanotic. He had a brassy tracheal cough and brought up about an ounce of frothy sputum per day. There was a pleural rub at the right base. Bronchoscopy by Dr. Fletcher revealed a frothy secretion around the right main bronchus, a reddish mass projected from the bronchus to the right middle lobe. The entrance to the right upper and right lower lobes was patent, though distorted. The septum between the right middle and right lower lobes ran diagonally from the right anteriorly to the left posteriorly. Bronchoscopy on January 27, 1925, showed that the granulating mass was smaller. The patient was in hospital from November 30, 1924, to February 27, 1925. He was given a pneumothorax for diagnostic purposes, but had no other treatment besides rest. On discharge he had sufficiently improved to go back to work. He reentered hospital several times with the same symptoms. On each entry dyspnea and cyanosis became more marked.

Bronchoscopy on October 14, 1927 revealed a red and injected tumor-like mass 35 cm. from the teeth. On January 14, 1930, nothing could be seen of the

granuloma, but the bronchial lumen was distorted, polygonal instead of round; no one bronchus was definitely stenosed. Below this lay the septum between the right middle and lower lobes. It was distorted and ran on a slant from the right anteriorly to the left posteriorly.

X-rays taken at intervals during the five years of observation constantly demonstrated an atelectatic right middle lobe.

CASE 24.—Stenosis right middle bronchus. Supportive peribronchial lymphadenitis. On September 24, 1928, Dr. C. C. Fulmer referred to me a housewife of forty-three, who at the age of eighteen had had a pneumonia that lasted a year, and who had been coughing but not expectorating ever since. The chest was tapped at the time but no fluid was found. She had no further febrile attacks up to four years ago, when she was operated upon for a calcified tumor of the uterus. After this she developed a double pneumonia and was very sick. An



Right Fig. 44 Left Fig. 45

Fig. 44.—Case 23. Stenosis of right middle lobe bronchus with atelectasis of lobe.
Fig. 45.—Same; lateral view.

abscess was said to have formed on the right side. Since this operation she had not only coughed but expectorated a cupful of sputum a day. The sputum had a sickly sweetish smell, but was not foul; it occasionally contained small flecks of blood. She had had pleuritic pain in the right chest for years, usually posteriorly; of late she had had pain under the right breast. For the last three years films of the chest had demonstrated a wedge-shaped shadow in the right chest corresponding to an atelectatic right middle lobe.

I found a well-nourished woman, whose fingers were not clubbed, but arthritic. The lower right chest was rather hyperresonant than otherwise. She had a barking cough, but no râles and no changes in breath sounds. On September 27, there was an area of dullness to the right of the sternum, but no râles.

On October 11, 1928, Dr. Harold Fletcher did a bronchoscopy, and reported as follows:

"The larynx and trachea are slightly injected. The main bronchus is markedly hyperemic. The membrane bleeds easily on pressure from the bronchoscope. About

2½ centimeters below the carina on the anteromedial wall of the main bronchus, there is a small mass of granulation tissue surrounding a fistulous opening from which a large amount of thick yellowish pus comes. A large amount of pus is removed by suction and an attempt made to evacuate the contents of this cavity by inserting a flexible suction tube. The opening was then moderately dilated with a Jackson's bougie."

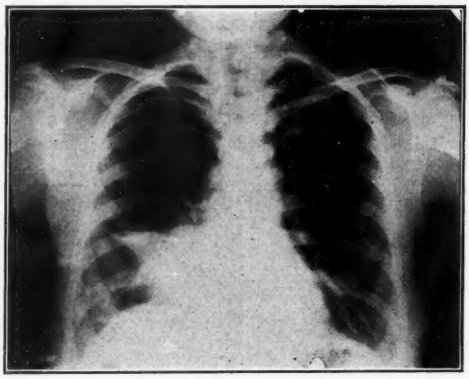


Fig. 46.—Case 24. Triangular shadow, right side of chest, corresponding to atelectatic middle lobe.

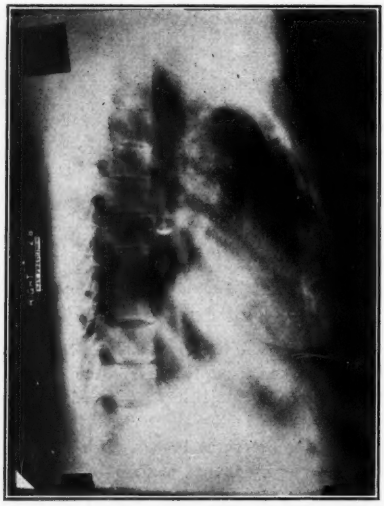


Fig. 47.—Same; lateral view.

Bronchoscopy was repeated on October 24. The right middle bronchus was obstructed. Once the obstruction was entered a coffee-cupful of pus was aspirated. Lipiodol was injected into sacculations of the stenotic right middle lobe.

Dr. Newell reported:

"Chest, after injection of lipiodol by bronchoscope.

"A very small quantity of lipiodol is seen in the right middle and lower lobes. Quite close to the hilum of the middle lobe this demonstrates some small cavities

about a centimeter in diameter, evidently definitely enlarged from the normal size of the bronchi there. There is so little lipiodol, however, that the details are not well brought out.

"The volume of the right middle lobe is considerably diminished and it is rather dense, especially near the hilum. The right diaphragm is high.

"*Conclusion:* These findings go well with the clinical diagnosis of bronchial gland suppuration with partial stenosis and chronic infection in the right middle lobe."

The sputum gave an almost pure growth of streptococcus viridans. No tubercle bacilli and no spirochetes were found in it and a guinea pig inoculated with it was negative. The patient objecting to a repetition of the bronchoscopy, the chest was opened on December 4, 1928, and a shrunken middle lobe exposed without opening the pleura. On enlarging this incision, however, a pneumothorax resulted. The middle lobe was so shrunken that its borders were made out with difficulty. It was firmly adherent to the very high diaphragm, the mediastinum and the pleura. The pneumothorax precluded the possibility of opening the lung, wherefore the air was aspirated from the pleura and the wound closed.

Second Operation: On December 19, 1928, the wound was reopened. The diaphragm was pulled very high. The pleura was perhaps one-eighth of an inch thick. The lobe was small and shrunken. It was pierced in several directions with a needle without encountering pus. It was carefully separated from the diaphragmatic surface, partly with the knife and partly with the finger until its front edge appeared. Upon severing this one encountered two small cavities filled with inspissated pus and these led into a larger one. From none of them did the patient blow out air, but a grooved director inserted into the larger one led toward the hilum. The cavity was packed with iodoform gauze and the wound left open.

The patient made a good recovery and has been considerably relieved.

Lipiodol is of great help. The technic of injection has been outlined above. By tilting the patient back and toward the affected side it is easy to make the oil flow into the lower bronchi; the right middle bronchus usually offers little difficulty if the patient leans forward and to the right. The upper bronchi, however, are hard to fill satisfactorily. Failure to inject them will usually leave a doubt whether an actual block or deficiencies of technic are at fault. When injection of the upper lobe is proposed the patient should be seated before the screen. Immediately the oil reaches the bifurcation he should be placed on the affected side, either horizontally or with the head slightly lowered. It may be easier to fill the upper lobe by cocainizing not only the pharynx but the larynx and inserting a fine rubber catheter through the nose into the trachea a little beyond the level of the bifurcation. With the patient horizontal and lying on the affected side, lipiodol is injected into the catheter. No time should be lost in exposing the film; the patient should not be allowed to sit up and should make every effort to keep from coughing until the films are developed. In upper lobe injections it is usually impracticable to follow the oil through before the screen; lower and middle lobe injections should be watched with the fluoroscope, which, as in the more central stenoses, often gives information missed in the films. One sees the oil flow down the trachea, enter the major bronchus, stop at the site of steno-

sis and after a quantity sufficient to fill the bronchus to its origin has been injected one sees it spill over and run down the next unobstructed bronchial opening. If the stenosis is incomplete a little oil may trickle slowly past and settle in distal bronchiectatic pouches. If the plain film reveals a shadow then films after lipiodol injection should be exposed sufficiently to penetrate the density covering the oil; an ordinary chest timing will be underexposed.

Localized stenosing processes of the upper air-ways are not hard to diagnose; physical, radiographic, and bronchoscopic evidence is distinctive. Much more difficult to recognize are *diffuse stenoses* of the *smaller bronchi.* Their symptoms are more characteristic than their

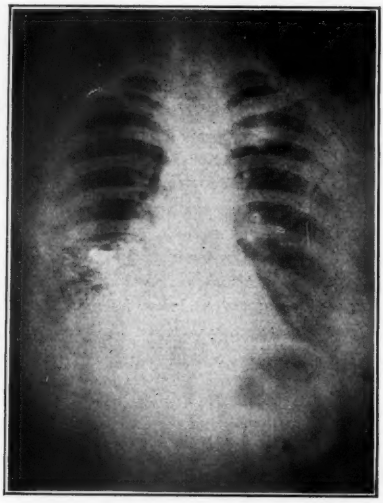


Fig. 48.—Bronchiectatic cavities distal to stenosis of right middle lobe, injected with lipiodol through bronchoscope.

signs. Constant dyspnea with alarming acute exacerbations without evidence of high obstruction arouses suspicion; examination, however, reveals few definitely confirmatory signs. The respiratory rate is often increased even during rest, the chest is flat rather than barrel-shaped, its excursions are small. The patient, especially during acute exacerbations, works hard to breathe; the intercostal spaces retract during inspiration but no one part of the chest retracts more than another. Toward the end when acute pulmonary dilatation supervenes the intercostal spaces may bulge. Vital capacity is much reduced. There may be some increase of interscapular dullness, usually not definite; nor is it likely that a deep-seated diffuse selerosing process would impair resonance. Often rather the resulting emphysema causes hyperresonance. Breath sounds may be everywhere diminished

in intensity. Squeaky dry musical ronchi and also coarse crepitant râles are scattered diffusely over the chest. The cough is often distressing; a frothy watery sputum, not like the scant sticky sputum of asthma is produced. It is sometimes purulent, a little blood tinged at times. Profuse hemoptysis, however, is not a part of the picture of diffuse peripheral stenosis. Often there may be signs of an old dry fibrous tuberculosis, but rarely of much peripheral induration or cavitation. The signs of emphysematous dilatation may be difficult to distinguish from cavities. Engorgement of the peripheral veins is not marked. Although the extremities may be markedly cyanotic, I have never seen them edematous. The pulse is small and rapid, thready at times. The blood pressure is low. The right heart is likely to be hypertrophied and at times dilated, although in theory it should be small. The bronchoscope may show sudden diffuse narrowing of the smaller bronchi; this, however, is a difficult thing to gauge.

The x-ray may show an increased hilar shadow and markedly increased diffuse peribronchial thickening; it may also show old pneumoconiotic deposits or old chalky or fibrous tubercles.

The diagnosis will usually rest on the cyanosis and constant dyspnea with acute exacerbations, unaccounted for by higher stenosis of the air passages or by cardiac or vascular disturbances. It will often remain obscure and doubtful until cleared by prolonged observation—and too frequently by autopsy.

Signs of still more peripheral stenosing processes, the *bronchiolitis fibrinosa* of Fraenkel[28] are, like those of diffuse stenosis of the smaller bronchi, indefinite. Here also breathing is rapid and shallow, there is some cyanosis, often hypertrophy of the right heart; and in the acute attacks of agonizing dyspnea an ashen pallor and signs of circulatory failure. Physical signs are those of emphysema, and perhaps during the acute attacks, of patchy bronchopneumonic consolidation. Bronchoscopically nothing but the diffuse frothy secretion is apparent. The enormous bubbles of peripheral emphysema seen in the x-ray are fairly characteristic. They are not infrequently mistaken for tubercular cavities.

DIAGNOSIS

Diagnosis of stenosis of the trachea and at the tracheal bifurcation is usually not difficult, given time and means to make it. It is important to distinguish lower stenosis from glottis edema and laryngeal obstruction, for tracheotomy while worse than useless in the one is imperative in the other. Gerhardt[7] considers the position of the head of diagnostic value. He states that in laryngeal stenosis the head is thrown back, in tracheal stenosis the chin is thrust forward and the head inclined a little downward. Respiratory excursion of the larynx is less in tracheal than laryngeal stenosis; the weight of the normal

atmospheric column of air with a vacuum underneath rests on the larynx in high stenosis, the pressure difference comes lower down in tracheal and other deep stenoses. Gerhardt lays much stress on this sign and states that if laryngeal excursion amounts to 1 cm. or less, the stenosis lies below the larynx. The voice is weak. Loud stridorous breathing is most clearly heard in the neck over the trachea. Laryngobronchoscopy affords an exact diagnosis. If there is the least doubt about the level of the stricture laryngoscopy or bronchoscopy should be tried very faithfully, before attempting tracheotomy. Lipiodol injection and x-ray will rarely be possible in an emergency but will help localize a chronic stenosis if laryngobronchoscopy cannot be done. Differentiation against diffuse low-lying peripheral stenosis is possible; in peripheral stenosis stridor is lacking, respiration is rapid and shallow, and both laryngobronchoscope and x-ray reveal unobstructed upper air passages.

Unilateral stenosis is recognized by unilateral impairment of respiratory excursion, retraction of the intercostal spaces, dullness or flatness and absence or weakness of breath sounds. These signs are easily mistaken for similar ones caused by fluid or a thick pleura. Retraction of the mediastinal viscera toward the affected side distinguishes the two; occasionally a heavy x-ray exposure will reveal detail through the grayness of an obstructed lung which so large an effusion would not show; an aspirating needle, if one cares to resort to this not wholly innocuous instrument gives a dry tap, and when attached to a manometer shows an intrathoracic pressure far below normal. Bronchoscopy again, will reveal the site of obstruction and sometimes its cause, and so will, usually, an x-ray picture with lipiodol. If the stenosis is accompanied by a pleural effusion diagnosis may be difficult.

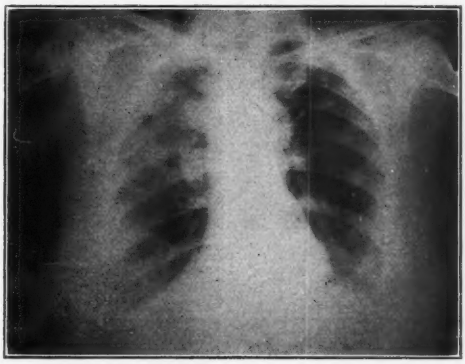
Complete stenosis involving one of the lobar bronchi or a large part of a lobe is distinguishable from other forms of consolidation by shrinkage of the lobe with retraction of the chest wall and viscera toward it. Frequently also by absence of expectoration, which sooner or later must appear in every pneumonic form of consolidation; uncomplicated stenosis is further distinguished by absence or irregularity of fever and other constitutional symptoms, and by the uniformity and density of the radiographic shadow. Retention of lipiodol at the site of stenosis and the bronchoscope confirm the diagnosis of stenosis in the lower and middle lobe. Time corroborates the suspicion—not necessarily a very long time—for no pneumonic consolidation lasts as long as a stenotic one without undergoing resolution, fibrosis or some change recognizable clinically and radiographically, in short, without something happening to it, while a stenotic consolidation may persist indefinitely. If the daily sputum is observed and measured a characteristic relation appears between intermission and recurrence of constitutional symptoms and expectoration; in pneumonic consolidation

rises in temperature do not ordinarily accompany cessation of expectoration.

Differentiation from an old chronic tubercular pneumonia may be extremely puzzling, especially in affections of the upper lobe, since both tuberculosis and stenosis cause shrinkage, fibrosis and cavitation. The presence of tubercle bacilli in the sputum or of tubercular lesions elsewhere may help. Often enough both tuberculosis and stenosis coexist and exact diagnosis may be impossible, unless continuous observation reveals the characteristic play of intermittent stenosis.

The following is a case illustrating the difficulties of decision:

CASE 25.—Tubercular stenosis right upper bronchus. A doctor's wife of fifty-three entered St. Joseph's Hospital in October, 1928, complaining of pain in the



Right Left

Fig. 49.—Case 25. Aug. 31, 1928: Density of right lung most marked at apex; cavitation at apex.

right chest and distressing paroxysms of cough, with a yellowish sputum which at times was offensive and often contained blood. She had had pleurisy with effusion twenty-five years ago. Three years previously and again in March, 1928, she had a cough and expectoration and was again in hospital. In May, 1928, she had a pneumonia with fever of 102°, cough and rusty sputum. She had bled at intervals since, usually after working in her garden. The sputum began to be offensive in August, 1928. She had a feeling that air did not enter the upper right chest when she breathed. She had lost twenty pounds in weight. X-rays taken in August at Eureka, California, showed a density with multiple cavities involving the right upper lobe. Films in October showed a large cavity, the lobe was dense and more shrunken; the smaller cavities had disappeared. The degree of density varied remarkably from one day to the next. The temperature was usually normal, occasionally reaching 100° F.

Physical examination showed a well-nourished woman. The right upper chest lagged. Physical signs over this area varied from day to day; it was sometimes dull, sometimes air-containing; the breath sounds were at times tubular, at times amphoric; at other times there were no cavity signs. The breath sounds were usually diminished in intensity and accompanied by râles. The right upper intercostal spaces were a little tender to pressure. The rest of the chest was normal. The

copious sputum was examined repeatedly, but no tubercle bacilli were ever found. Dr. Ophüls examined seven specimens for tumor cells; none were found.

Dr. Rehfisch reported x-ray examination of October 22, 1928, as follows:

"There is a lobar consolidation involving the right upper lobe with slight displacement of the trachea toward the right. There is a definite area of cavitation in the center of the consolidated parenchyma and some coarse, mottled, linear

Fig. 50 Fig. 51

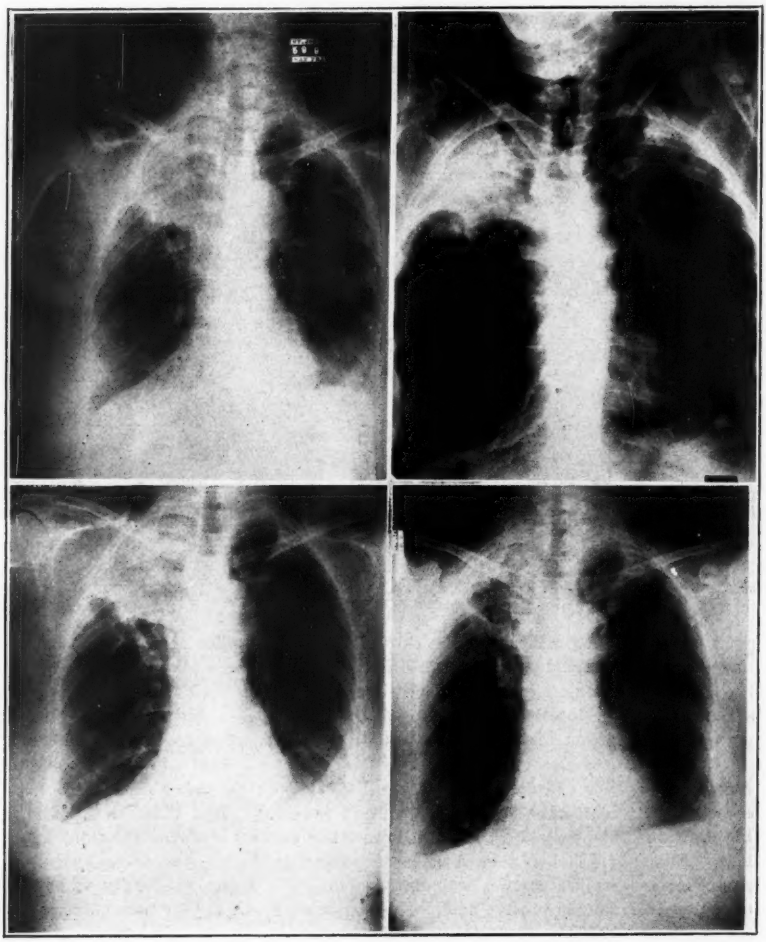


Fig. 52 Fig. 53

Fig. 50.—Oct. 22, 1928: Moderate density of right upper lobe. (For legend see Fig. 52.)

Fig. 51.—Oct. 25, 1928: Marked density, left upper lobe. (For legend see Fig. 52.)

Fig. 52.—Oct. 26, 1928: Left upper lobe less dense. The variation in films taken a day or two apart (Figs. 50, 51 and 52) demonstrates that the grayness is due to a varying air content rather than to a tubercular infiltrate.

Fig. 53.—Case 25. January 8, 1929: Right apex is much clearer; trachea is retracted to right and right upper lobe shrunken.

shadows running downward from its lower border toward the lung root. There are a few small calcified foci at the left apex but no evidence of active pathology on that side.

"The heart and aorta are within normal limits of size and shape. *Conclusion:* Pulmonary consolidation, right upper lobe, with necrosis in the middle of that lobe.

"From a purely x-ray point of view an infectious process, of which the most likely is tuberculosis, would seem to be the most probable etiologic agent. At the same time it is impossible to rule out some bronchostenotic process and a bronchial tumor must at least be considered. Clinical correlation would be unusually helpful in this case."

Dr. Chamberlain made the following report on these films:

"There is a dense area at the top of the right lung field which can only be explained on a basis of atelectasis of the right upper lobe. The lobe is shrunken to perhaps a fifth of its normal volume, but the interlobar septum can still be made out both in the lateral and in the anterior projections. There is very little detail to be made out in the atelectatic lobe. One of the earlier films, taken elsewhere, shows a cavity in the affected lobe, but this is not seen in our films.

"In the left lung apex there is a little granular density which I consider quite characteristic of old tuberculosis scars.

"*Conclusion:* A complete or almost complete closure of the right upper lobe bronchus is demonstrated by these findings, and it is hardly likely that the cause of this is anything else except a primary carcinoma of the bronchus. The only other possibility which deserves serious consideration would be an old shrinkage of the right upper lobe, result of tuberculosis of many years' standing. I do not think that an upper lobe abscess with granulations closing the bronchus would produce such a small lobe. On account of the unlikelihood of tuberculosis or abscess producing this picture, I feel that it is almost surely a carcinoma."

Dr. Lloyd Bryan, who was kind enough to examine these films, as well as films after lipiodol injection taken on November 5, made the following report:

"The lipiodol injection demonstrates that there is a rounded irregular shadow at the root of the upper right bronchus which is pressing upon the bronchus giving a partial stenosis. The films taken at this time show considerably more air in the upper right lobe than in the film taken previously. While primary malignancy cannot be definitely excluded, it seems to me that the whole picture can be explained on the basis of a partial stenosis from pressure of a gland upon the bronchus. There is definite evidence of old tuberculosis at both apices, pleuritis at both bases, and mediastinal pleuritis on the right side. While the presence of tuberculosis does not exclude the possibility of carcinoma, it seems to me that the whole picture can be explained on the basis of an old tuberculosis plus glandular involvement."

On October 24, Dr. Harold Fletcher did a bronchoscopy and reported as follows:

"A great deal of difficulty is encountered in inserting the bronchoscope past the larynx because of lack of cooperation on the part of the patient. Bifurcation of trachea negative except that the lateral wall of the right main bronchus was much more concave than normal, making the location of the opening of the upper right lobe of the bronchus difficult to find. To locate the opening of the upper right lobe of the bronchus, it was necessary to force the patient's head far to the left exerting pressure on the lateral wall of the right main bronchus. The opening was apparently normal in position, but the membrane lining this opening appeared definitely edematous, markedly pale and somewhat glairy. Apparently very little air entered or came out of this upper lobe. This last finding was indefinite. There was a small amount of pus just inside the entrance of the upper right lobe bronchus. No definite obstruction could be seen in it, but the impression given by this

examination was that there was an obstruction beyond the opening, probably coming from pressure above the upper right lobe bronchus and pushing somewhat in on the lateral wall of the right main bronchus, above the bifurcation of the upper right lobe bronchus. This last finding also must be put down as an impression rather than an actual proved condition."

It was difficult to decide between intrinsic stenosis; i.e., a tumor, and extrinsic stenosis from pressure of a hilar gland. It was also difficult to decide whether to explore the chest or to observe the patient further. Finally, however, the prolonged history of recurrent attacks, the presence of old spots at the left apex, the mediastinal thickening below and the definite evidence of basal pleurisy, as well as the concavity which forced the bronchoscopist to turn his instrument to the left before visualizing the right upper bronchus, made the likelihood of enlarged hilar glands sufficient to lay the question of tumor open to considerable doubt. It was decided to defer operation. The subsequent course justified the decision, for the patient has continued to improve in the past two years.

Distinction between tubercular and tumor stenosis is of much practical importance. Lipiodol and the bronchoscope will probably solve the problem in affections of the middle and lower lobes. In the upper lobe they fail. Cavitation and shrinkage with sputum persistently free from tubercle bacilli, absence of tuberculosis elsewhere, speak for nontubercular stenosis; so do repeated sharp rises in temperature of short duration, coincidental with increased dullness, cessation of expectoration and increased density on the x-ray film. When definite evidence, such as metastatic malignant glands or tubercular sputum is wanting diagnosis requires time and persistent observation. Evidence speaking strongly for tumor, however, makes time precious and then exploratory thoracotomy will need be considered.

Aneurysm may be difficult to distinguish from upper lobe stenosis, and if the aneurysm itself produces a stenosis, impossible. A pulsating shadow, evidence of syphilis, a bruit, a tracheal tug may help, but, notoriously, all these signs may be absent in aneurysm of the descending aorta. Continued observation and repeated x-rays may detect a free interval with remission of stenotic signs and films taken at so happy a period may show an aneurysmal shadow separate from the grayness of a previously stenotic lobe.

A brassy, barking, uncontrollable, nonproductive cough is characteristic of mediastinal and tracheal irritation and is common to various stenosing lesions of the trachea, the bifurcation and adjacent portions of the bronchial tree. The more distant the lesion from the midline the less likely is this characteristic cough to be a prominent symptom. Cough then is usually troublesome only in those unilobar obstructions which have begun to encroach upon the hilum or which from the beginning lie close to it. It is rarely so loud or persistent as in more central stenoses. Interlobar pleurisy and other encapsulated collections of fluid may simulate lobar stenosis. Very old ones may even draw the mediastinum toward them. Partially communicating collections of pus, abscesses and empyemas, in which the communication

becomes blocked may simulate intermittent stenosis; fever may accompany cessation of expectoration and defervescence increase of expectoration.

In such cases examination of the sputum will help. The sputum of a perforating empyema consists almost entirely of pus; pus containing a single strain of organisms, streptococci or staphylococci, without contamination of anaerobes derived from the air passages is characteristic. Sputum of recently perforated abscess is usually blood stained, has a characteristic stench and often contains elastic fibers and shreds of lung tissue. The lung tissue is sometimes visible to the naked eye as grey, putty-colored, or black shreds and particles. The bacterial flora is immensely varied and often includes spirillae and fusiform bacilli. Sputum of a stenosis may be blood stained, but is often frothy and watery especially immediately following an intermission. It often separates into three layers; it is less likely to contain elastic fibers and elements derived from destruction of lung tissue. Its flora, while varied, is less likely to contain spirillae, fusiform bacilli, and other spore-bearing anaerobes.

The aspirating needle may draw pus from a localized empyema, and exclusion of a stenosis by means of the bronchoscope and lipiodol may lead to a definite conclusion.

Cysts, dermoids, carcinomas and other intrathoracic growths may occasionally give the physical findings of stenosis and their radiographic shadow may simulate that of a collapsed lobe. Here again, mediastinal retraction is wanting and the bronchoscope and lipiodol will serve to exclude stenosis.

Recognition of the underlying causes of obstruction of the major bronchi not only more accurately delineates the various kinds of obstruction but serves to separate the stenoses from other disorders with which they may be confused. Signs of syphilis and a positive Wassermann reaction may accompany syphilitic stenosis, localized usually in the trachea and at or near the bifurcation. Signs of syphilis, combined with those of aneurysm of the descending aorta may lead to recognition of left upper lobe stenosis or tracheal stenosis; so may pupillary differences and hoarseness due to recurrent laryngeal palsy, similarly signs of aneurysm of the ascending aorta may explain a right upper bronchial stenosis. Signs of pulmonary tuberculosis elsewhere are to be sought for, especially tubercular adenopathy—glands in the axilla or above the clavicles. Tubercle bacilli in the sputum, a positive v. Pirquet reaction, a low white cell count with lymphocytosis and other auxiliary signs are of value.

Premonitory hemoptysis without other signs referable to the respiratory tract and without impairment of general health often presages tumor in older persons. Metastatic axillary or supraclavicular glands, bony metastases in the ribs or vertebrae may occasionally verify such

a suspicion. Unexplained distant venous thromboses in the femoral or iliac veins or the superior vena cava are sometimes among the first signs of a lung tumor.

A history of exposure to dust and demonstration of pneumoconiotic deposits speak for nontubercular deforming peribronchial inflammation. Disturbances of deglutition, mediastinal pleurisy, mediastinal x-ray shadows, and distortion of the esophagus will add confirmatory evidence if intraesophageal lesions are excluded.

Diagnosis of diffuse peripheral obstruction may be a matter of great difficulty and require prolonged observation. Diagnosis will be possible mainly by exclusion. It will rest mainly upon cyanosis and constant otherwise unexplained dyspnea interspersed by acute exacerbations. There may be a past history of dusty trades or of tuberculosis. The physical findings are not characteristic; bronchoscopy and x-ray with and without lipiodol rarely give information positive enough to establish a diagnosis. Asthma and extrapulmonary causes of dyspnea, especially cardiovascular derangements, must be carefully excluded. A long prodromal history and the nature of the attacks themselves may give a clue. The attacks are distinguishable from true asthma, for the dyspnea is not purely expiratory, and not spasmodic but more or less constant, there is not so much wheezing, the patients are not florid and plethoric, their chests are not barrel-shaped, but flat. The intercostal spaces may be markedly retracted, but during an attack, they probably bulge, for the lungs are found distended at autopsy. Subcutaneous emphysema has been observed.

The fibrinous bronchiolitis of Fraenkel[28] which differs from the foregoing only in its acuteness and severity is due to an essentially similar stenosis; a history of inhalation of destructive fumes or gases, marked collapse, frothy or bloody sputum with agonizing dyspnea, will serve to distinguish it. The dyspnea may differ from that of stenosis of the smaller bronchi in being more expiratory than inspiratory. In two recent cases of stenosis of smaller bronchi the chest was distinctly flat, and the intercostal spaces retracted, although at one autopsy the lung was greatly dilated. Fraenkel, however, mentions expiratory dyspnea and dilatation of the lung so severe as to have caused subcutaneous emphysema in one of his patients.

(To be concluded in June number.)

BRONCHIECTASIS*

(To be published serially in four installments)

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(Continued from p. 326)

III. TREATMENT

THE various forms of treatment may be divided into two groups. These are (1) nonoperative and (2) operative. But before considering them let us point out that a *cure can only be considered when the diseased portion of the lung has been removed or destroyed.* Most forms of treatment only influence symptoms. And, in this connection, it is well to remember that the size and shape of the dilatations are seldom influenced by these forms of treatment. One must, therefore, be cautious about evaluating the results obtained by those forms of treatment which do not destroy the diseased bronchi and lung tissue. It should also be appreciated that bronchiectasis is characterized by remissions and that striking early improvement may later be nullified. More than a symptomatic cure can hardly be expected in most cases. On some occasions relief from symptoms is apparently all that is necessary. Little evidence has been forthcoming so far to indicate that spontaneous cures occur very frequently. Still the condition need not progress in every case. The age of the patient, mode of origin, nature of lesion, presence or absence of local or general complications are all important considerations. A bilateral lesion obviously increases the hazard, and we agree with Ewart, who stated that mere fetor is not in itself an alarming sign.

A. NONOPERATIVE PROCEDURES EMPLOYED IN THE TREATMENT OF BRONCHIECTASIS

I. Rest. II. Diet. III. Climate.
IV. Postural drainage. V. Thirst cure. VI. Heliotherapy.
VII. Intravenous therapy—neosalvarsan, colloidal silver.
VIII. Direct intrabronchial application of drugs by syringe or spray.
IX. Irrigation of the lung (bronchial lavage).
X. Inhalations of superheated air with a mixture of suitable drugs.
XI. Vaccine therapy.

I. REST

Bronchiectasis is a disease which seems to be characterized by remissions and seasonal variations during which time the patient may be entirely free from cough and expectoration. This possibility must always be borne in mind when offering

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a prognosis.' Rest in bed usually gives relief even with marked diminution of the sputum but never any lasting improvement after the patient gets up.

II. DIET

All forms of diet including those rich in vitamins have been employed. None of them have given worth while results.

III. CLIMATE

There are many who recommend the same sort of climate to patients suffering from bronchiectasis as they do to patients suffering from tuberculosis. There is little evidence at the present time to indicate that a change in climate is of any definite value in such cases, although it is advisable for a patient with bronchiectasis to avoid atmosphere which contains irritating gases.

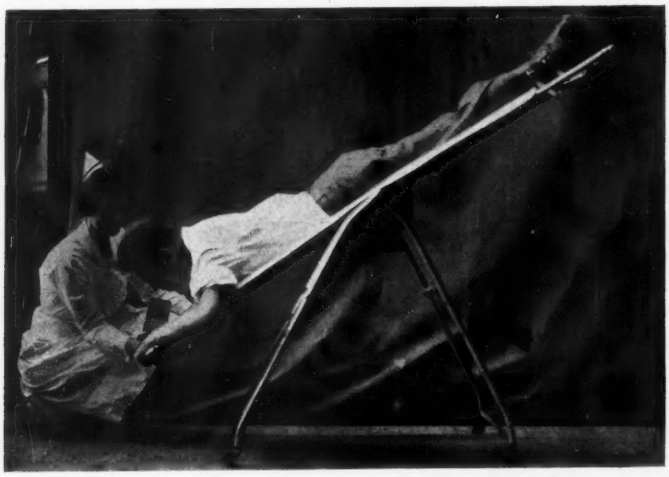


Fig. 21.—Postural drainage table. (Singer-Phillips.)

IV. POSTURAL DRAINAGE

Postural drainage is most valuable. This method may be carried out continuously or may be employed intermittently (Fig. 21). The purpose of continuous postural drainage is to have the patient carry on this form of treatment both by day and by night. Continuous postural treatment is carried out by elevating the foot of the bed. Various devices have been recommended. Singer has recently devised a bed which can be used for continuous or intermittent postural drainage.* One can employ a modified Quincke tilting table for intermittent postural drainage. Many patients suffering from mild bronchiectasis require no other form of treatment. They are able to go about their work if they empty their dilatations early in the morning. Some are required to repeat this postural drainage several times during the day. Intermittent postural drainage should never exceed two or three minutes at a

*A description of this bed is now in press in this journal.

time and may be carried out with the patient lying either on his abdomen or back. Postural drainage should always be given a fair trial before any other form of treatment is suggested. In certain cases it can be supplemented with profit by bronchoscopic aspirations.

According to some, expectorants constitute an important adjunct to postural treatment and may also be employed in combination with drugs which are instilled directly into the tracheobronchial tract. Ewart was convinced that it was wise to wash out the bronchi with their own secretions and that the use of expectorants was not contraindicated even though they undoubtedly increased secretions. He described the following procedure in 1901. The first step was to secure free drainage by posture as tested by the thermometer. In other words, a drop in the temperature curve was to be expected if the drainage was effective. The second step in the procedure was to set up a slight drug catarrh in order to clear away if possible the causes of fetor. This was accomplished by the administration of potassium iodide. The third and final step was to "squeeze out the secretion" by emetics after three or four days. It was Ewart's opinion that vomiting appeared to contract the muscularis mucosae in a degree not obtainable by simple cough. Unfortunately the thermometer seldom shows any changes following postural drainage. Some patients with bronchiectasis do feel better following the use of expectorants, but no one substance is completely effective in the treatment of fetor. Creosote has been employed. We have observed some diminution in the amount of fetor following the intravenous administration of neosalvarsan. The production of adequate drainage is still the most effective means we have of treating fetor.

V. THIRST CURE

It was once thought that a reduction in the fluid intake might result in a reduction in the amount of expectoration since the latter, in the case of bronchiectasis, is to a very large extent composed of water. This form of treatment has been given a fair trial and has been shown to be of little or no value.

VI. HELIOTHERAPY

The use of sunlight, quartz light, etc., has no particular value in the treatment.

VII. INTRAVENOUS THERAPY

Several drugs have been employed intravenously in the treatment of bronchiectasis. Colloidal silver found favor for a time. At the present time neoarsphenamine is perhaps the only drug given intravenously. It is used when the sputum contains many fusiform bacilli and spirochetes. The therapeutic results are sometimes striking. This is usually the case when there is adequate drainage and little retention of secretions. Unfortunately, many patients with bronchiectasis whose sputum contains the above-mentioned organisms fail to respond to this form of treatment. *We have never observed a patient with definite bronchiectasis as demonstrated by lipiodol in whom by the use of neosalvarsan alone there was accomplished either a permanent subsidence of the symptoms or a diminution in the size of the bronchial dilatations.* This form of treatment may be helpful, however, in combination with other forms of treatment. (See previous discussion on bronchiectasis and fusospirochetal disease.)

VIII. DIRECT INTRABRONCHIAL APPLICATION OF DRUGS BY SYRINGE OR SPRAY

For years many drugs have been injected into the bronchial tree not only in the treatment of bronchitis and tuberculosis but also bronchiectasis. At the present time lipiodol is frequently used. Lipiodol has no definite bactericidal properties. Neuswanger found when agar plates, upon which iodized oil was smeared, were inoculated with B. coli, Staphylococcus aureus and Streptococcus hemolyticus that the organisms were found growing in close proximity to the globules of oil and that no inhibitory action was observed. However, quantities of the oil exposed to the air for several months showed no evidence of degeneration; and no colonies of bacteria or fungi were observed. Archibald and Brown performed similar experiments and came to the same conclusions. They found that 3 c.c. of lipiodol mixed with an equal quantity of thick tenacious sputum taken from a patient with bronchiectasis kept at room temperature for forty-eight hours and then streaked on various culture media and incubated gave a luxuriant growth which included pseudodiphtheroids, pneumococci, staphylococci and micrococcus catarrhalis.

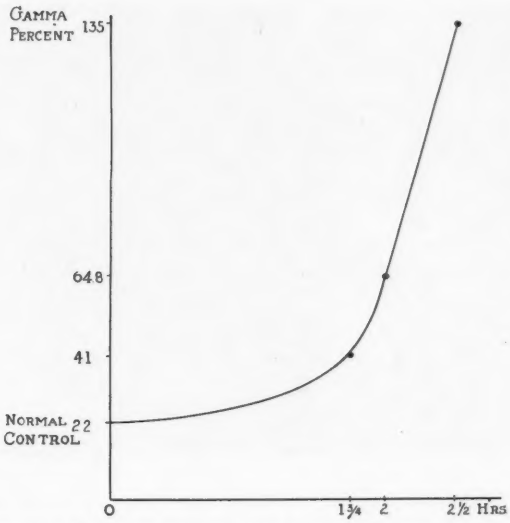
It has been shown experimentally by one of us (H. C. B.) that lipiodol may remain unchanged in normal lungs for weeks or months. There is no lymphatic distention and hence no subsequent infiltration or fibrosis. Mononuclear cells engulfed with oil are not infrequently to be seen in the aveoli. Reference has already been made to the question of expectoration time as determined by lipiodol.

IS THE IODINE IN LIPIODOL OF SPECIFIC THERAPEUTIC VALUE?

One might perhaps ask whether the iodine content (40 per cent) of lipiodol plays a significant part. The work of Cole and Womack has shown quite clearly that infection reduces the iodine content of the thyroid gland. Loeb and Michaud and others have shown that tuberculous tissue, and all inflammatory foci have a high affinity for iodine and store it in abnormally high amounts. The iodine in lipiodol is intimately bound up with the poppy seed oil. Moller and Magnus state that examination of urine following lipiodol injection showed that the first trace of iodine appears about six hours after injection. The excretion then rapidly increases, the maximum being reached in about twenty-four hours. The rate of excretion was then observed to slow up considerably. In their experiments they found no trace of iodine in the urine after six days. This corresponds to the sequence of events when iodine preparations are administered orally (Sturm).

C. B. Davis and George M. Curtis, among others, have pointed out the precautions necessary for the accurate quantitative determination of the iodine content of blood. Such methods call for the service of a trained chemist. In order to obtain accurate information as to the passage of iodine into the blood stream following the intrapulmonary injection of lipiodol, Curtis, Cole and Ballon injected 5 c.c. of lipiodol into the tracheobronchial tract of two dogs, having previously ligated the esophagus and cut across and isolated the trachea. The ligation of the esophagus was essential since any lipiodol which might have escaped into the gastrointestinal tract would, of course, have led to erroneous conclusions. Absorption could also take place from the pharyngeal

mucosa. The blood iodine determinations in these two experiments have shown the following: (see Curve 1 for the result of the first experiment.) The blood iodine in the second dog rose from 22 gamma* per cent (normal control) to 81.2 gamma per cent after two and one-half hours. After three hours the determination obtained was 77.8 gamma per cent. It is thus evident that iodine is rapidly absorbed from the intrapulmonary lipiodol since the blood iodine rises from 3.5 to 6 times the normal control in two and one-half hours. Similar rises occur after oral administration of iodine preparations. It is hardly necessary to mention that many patients swallow some of the lipiodol during the course of the injection.



Curve 1.—Curve 1 shows the rise in blood iodine following the intratracheal injection of 5 c.c. of lipiodol into a normal dog weighing 9 kilo. This dog (Experiment 1) received 4.5 c.c. (0.45 gms.) of amytal intraperitoneally. The normal control was 22 gamma per cent. (Curtis, Cole, Ballon.)

The distribution of the dilatations remains unchanged following the therapeutic administration of lipiodol and at the present time it seems to be most helpful in the less advanced forms of cylindrical bronchiectasis.

Some patients undoubtedly feel better following these injections. Our results, however, have not been nearly so striking as those recorded by Ochsner. That lipiodol is of some therapeutic value has also been noted by Lenk and Haslinger, Sgalitzer, D. H. Ballon and others. The latter is of the opinion that the beneficial results are due practically entirely to the fact that the lipiodol can displace the sputum. Ochsner treated 112 patients with bronchiectasis with repeated injections of iodized oil into the tracheobronchial tree. The largest number of fillings received by any one patient did not exceed sixteen. In 35 (32 per cent) there was

*One gamma equals 0.001 mg.

complete relief of all symptoms and in 4 cases not only was the relief symptomatic but apparently the dilatations disappeared. "In 41 cases (36 per cent) symptomatic relief was obtained, but following an acute respiratory infection there was a return of some cough with a slight amount of sputum." Improvement was marked in the remaining 36 (32 per cent).

Singer has treated some patients in this manner with weekly refills for almost two years (Fig. 22). The only visible untoward effect seems to be the drowning of certain portions of the lung. Of greater importance, however, is the fact that none of his patients show the striking beneficial results just referred to. If the patients are observed long enough then one will find that they will have acute exacerbations. We

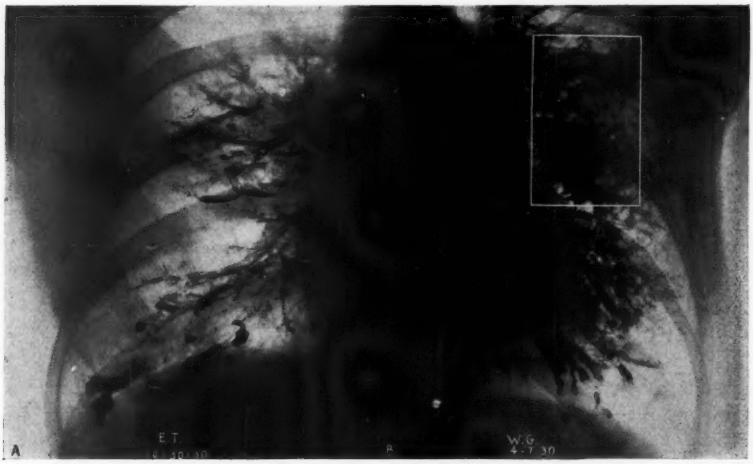


Fig. 22.—Lipiodol in the treatment of bronchiectasis.
A. (E. T.) concerns a male aged thirty-eight with what appears to be a combination of congenital (indicated by arrow) and acquired bronchiectasis. The chief complaints in this case, present for at least eighteen years, were hemoptysis, cough and foul expectoration. He also had bilateral maxillary sinusitis. This patient obtained some relief from the use of autogenous vaccines and potassium iodide. He also received one bronchoscopic treatment. Postural drainage benefited him most. Therapeutic lipiodol injections were given at weekly intervals for more than four months. This form of treatment reduced the patient's expectoration slightly but the physical signs and dilatations remained as before. The combination of postural drainage and lipiodol injection seemed to improve his fetor.
B. (W. G.) aged thirty-two, has bilateral bronchiectasis of at least fourteen years' duration. Treatment of his nasal sinus infection improved his cough and expectoration. This patient received therapeutic lipiodol injections at weekly intervals for almost two years with moderate improvement. The only visible untoward effect seems to be the "drowning" of certain portions of the lung (indicated by the rectangle).

have never observed the disappearance of dilatations and at the present time cannot admit that the use of lipiodol in the manner described has resulted in any lasting clinical cure of any of our patients with true bronchiectasis. We feel, too, that the period of observations must exceed several years. The number of injections must dispel any suggestion that the relief was not a mere coincidence; or that the patient was suffering from an ordinary superimposed acute upper respiratory tract infection which was making otherwise dry dilatations wet. The bene-

ficial effect of the iodine per se is problematic since the physiologic state of the thyroid gland is unknown.

IX. BRONCHIAL IRRIGATION (LAVAGE)

This method of treating pulmonary suppuration was first suggested by Yankauer in 1914. Although the irrigations were originally carried out through the bronchoscope there are some who employ a soft rubber Coudé catheter (Stitt and Wooding). The latter employ a hypertonic solution of saline and have reported 35 per cent symptomatic cures in a series of 100 cases. There was one death. Many bronchoscopists have abandoned this form of treatment not only because they observed reactions but chiefly because they concluded that the aspiration of bronchial secretions suffices. The injection of iodized oil accomplishes the same purpose. We prefer postural drainage and bronchoscopic aspiration whenever possible.

X. INHALATIONS OF SUPERHEATED AIR WITH A MIXTURE OF SUITABLE DRUGS

The use of inhalations of superheated air with a mixture of suitable drugs such as eucalyptus oil, etc., as a means of treating bronchiectasis has been discarded.

XI. VACCINE THERAPY

It is more or less agreed that vaccine therapy is of no particular value in the treatment of bronchiectasis. A few striking improvements are recorded in the literature. Jacquet, Roquejeoffre and Alavoine reported that the bronchorrhea and arthritis of a patient with bronchiectasis and arthritis had improved following vaccine therapy.

B. Operative Procedures

XII. Bronchoscopy.
XIII. Pneumothorax.
XIV. Oleothorax.
XV. Compression by means of pack and plombage.
XVI. Thoracoplasty.
XVII. Phrenicectomy.
XVIII. Ligation of the pulmonary artery.
XIX. Pneumotomy.
XX. Cautery pneumectomy.
XXI. Lobectomy.

XII. BRONCHOSCOPY IN THE TREATMENT OF BRONCHIECTASIS

From what has been said one cannot help but feel that mechanical factors and chronic bronchitis play important parts in the production of bronchiectasis. Chevalier Jackson has shown that in the bronchi there may be (1) stop valve obstruction in which no air passes in or out of the tributary area of the lung, (2) that there may be by-pass valve obstruction in which a diminished amount of air passes both in and out, and, (3) check valve obstruction in which the air is permitted to pass one way only. Emphysema, atelectasis, pulmonary fibrosis and bronchiectasis may be end-results. Internal drainage, that is, the spilling over of secretions from one bronchus to another which occasionally results in abscess formation, is also an important consideration.

The value of the bronchoscope in the treatment of foreign body bronchiectasis and in certain cases which are due to partial bronchial stenosis is obvious. The chief object in the treatment of most cases of bronchiectasis is to promote drainage. Postural drainage satisfactorily

accomplishes this in many cases, but it is doubtful whether postural drainage can ever as completely empty a dilated bronchus as bronchoscopic aspiration. To verify this fact one has merely to inject the lungs of a patient suffering from bronchiectasis by passive methods after postural drainage and by the bronchoscopic method after bronchoscopic aspiration. The latter usually gives a more nearly complete picture of the underlying condition. *Neither postural drainage nor bronchoscopy will in the strict sense of the term cure many patients with bronchiectasis, but they can cause arrest* of symptoms in certain cases. The important consideration is not the dilatation but whether it is wet or dry.

Unfortunately up to the present time most reports concerning the value of the bronchoscopic treatment of bronchiectasis have concerned themselves merely with stating whether or not the lesion was unilateral or bilateral and in the reported cases have included forms associated with multiple abscess formation and with pneumonitis. The lipiodol injection pictures have usually not been recorded. The value of bronchoscopy in the treatment of bronchiectasis lies therefore in removing or treating local causes (foreign bodies, bronchial stones, dilating strictures), in promoting drainage, and in attempting to prevent the progression of the disease (spilling over of secretions). Perhaps its greatest value may some day be in the treatment of those conditions which may lead to bronchiectasis. Clerf and D. H. Ballon believe that the milder forms of bronchiectasis associated with nasal sinus suppuration should be treated bronchoscopically. Disorders of the nasal sinuses should of course be corrected.

G. Ewart Martin, in 1929, reported that he had examined and treated 61 patients suffering from bronchiectasis. The ages of the patients varied from five and one-half to fifty-seven years. The lesion was bilateral on sixteen occasions. No mention is made of lipiodol injections. Two patients were completely relieved after one bronchoscopy with aspiration and lavage; one patient after two such treatments; two after three such treatments; one after five, and one after ten treatments. There were in all fifteen clinical cures. Two patients who could have been included in this group died of other complaints. There were eleven other deaths. Four patients ceased treatments and twenty-nine are still under observation. One of the patients with right-sided bronchiectasis reported to have died of other complaints developed a lobar pneumonia in the opposite lung. One patient died of encephalitis lethargica, one of brain abscess, one of uremia and three of influenza. It is fair to assume that practically all the patients reported as dead died from some of the consequences of the disease. Martin referred to the combination of bronchoscopic aspiration and pneumothorax but makes no mention of its failure. He is apparently of the opinion that bronchoscopic aspiration can, with profit, be combined with lavage.

It is admitted from the start that no method such as bronchoscopy will ever cure a patient with advanced bronchiectasis, but it is equally certain that many with advanced bronchiectasis may be rendered fit members of society.

XIII. TREATMENT OF BRONCHIECTASIS BY MEANS OF PNEUMOTHORAX

The treatment of bronchiectasis by pneumothorax is a subject which has been under discussion since 1903. Tillman in 1923 was able to collect 65 cases. It is at once appreciated that the value of these statistics is not great since lipiodol injections were not employed in these cases. But few cases have been added to the literature since 1923. Sixty-one per cent of the cases referred to above were unilateral, 18 per cent were bilateral. The condition was noted 27 times in males, and 16 times in females; the sex was not given in 22. The left side was involved 18 times in the unilateral cases, the right side on 10 occasions. The side affected was not recorded on 12 occasions.

DURATION OF ILLNESS BEFORE PNEUMOTHORAX WAS COMMENCED (*Tillman*)

Less than 1 year	1-3 years	3-5 years	Over 5 years	Period not known
3	14	7	16	25

The degree of bronchiectasis was mild in 4 cases, moderately severe in 2, severe in 11 and unknown in 28. Unsuccessful pneumothorax results were obtained in 21, this in spite of the fact that partial pneumothorax was possible in 11 of the 21 cases. In our experience pneumothorax is possible in about 6 out of every 10 attempted cases, but it can be maintained for more than six months in about 1 out of every 10 cases.

The duration of the treatment on the basis of 44 cases (Tillman) was as follows: 6 months, 17 cases; 6 to 12 months, 8 cases; over one year, 7 cases; unknown, 12 cases. The positive direct results in the 44 cases were as follows: Slightly improved, 11 cases; moderately improved, 17 cases; definitely improved, 16 cases. The direct results varied according to the duration of the illness. There were fewer definite improvements in cases of over three years' duration and more definite improvement in cases in which the history of illness did not exceed three years.

Tillman has classified the positive results in 44 cases according to the intensity of the disease as follows:

INTENSITY	SLIGHTLY IMPROVED	MODERATELY IMPROVED	DEFINITELY IMPROVED
Severe	1	1	1
Moderate	4	7	9
Slight	1	6	1
Unknown	5	3	5

As far as we are aware no large series of cases of bronchiectasis in which lipiodol injections had been performed have been reported. We therefore present the following cases (Fig. 23) drawn from a rather large experience. They suffice to substantiate the statement previously made by Brauer that he had never seen a patient with proved bronchiectasis cured by pneumothorax therapy. From the above it is certain that very *few patients with true bronchiectasis respond to pneumothorax treatment even though collapse may be almost complete.* Fig. 23, *7* illustrates the fact that a complete pneumothorax may compress everything but the dilatations. One might expect that when the dilatations extend well to the periphery the results might be better than when the dilatations are confined to the paravertebral area. Unfortunately this is seldom the case. We have observed, however, that the pneumothorax treatment may change the position of the lobe so that the dilatations become horizontal instead of vertical. Better drainage

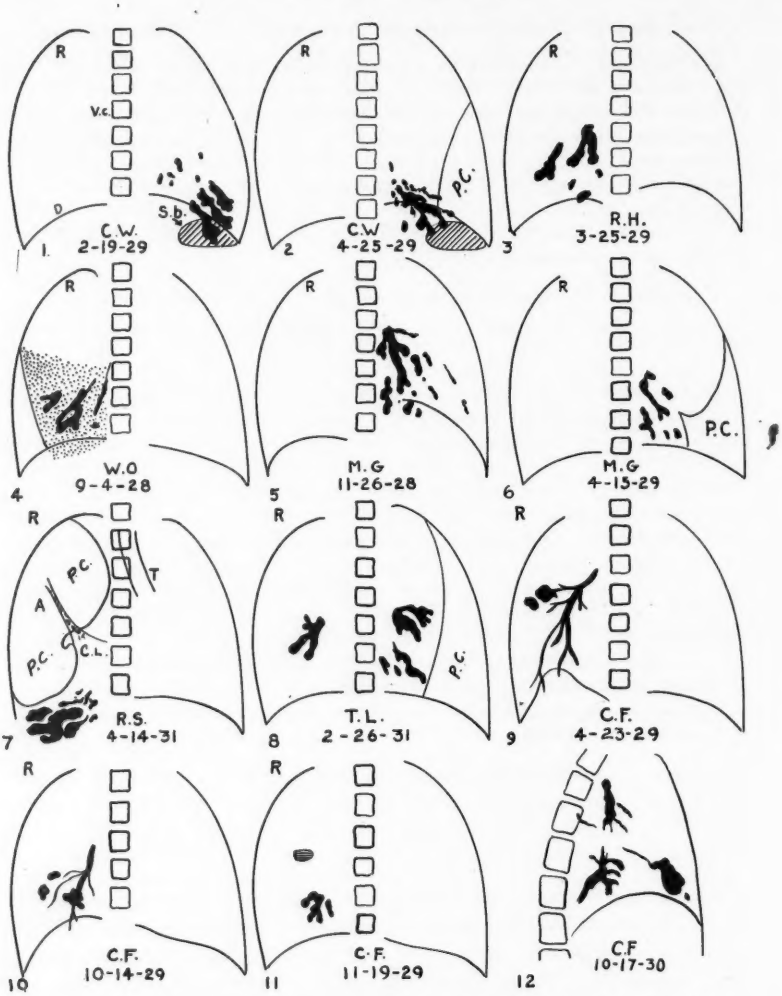


Fig. 23.—The above sketches are exact reproductions from the films (lipiodol injections) of patients with bronchiectasis who received pneumothorax treatments. (V. c. = vertebral column; S. b. = stomach bubble; D. = diaphragm; P. c. = pneumothorax cavity.)

Figs. *1* and *2* concern a male, aged eighteen, who suffered from left lower lobe bronchiectasis due to influenza (1918). The dilatations in this case extended well to the periphery. A phrenicectomy seemed to cause rotation of the bronchi (Fig. *2*). This patient received pneumothorax treatment. He also failed to respond to the intravenous administration of neosalvarsan. The pneumothorax treatments were carried on for about eight months. This patient died suddenly following such a treatment elsewhere. The cause of death is uncertain. A spontaneous or tension pneumothorax and mediastinal emphysema were considered.

Fig. *3* concerns a female, aged twenty, with right-sided postpneumonic bronchiectasis of several years' duration. Her nasal sinuses were treated with only slight benefit. A phrenicectomy was performed on November 7, 1929. The diaphragm failed to rise, the patient's condition was not improved. This patient received pneumothorax treatment for the better part of a year. It was possible to obtain almost complete collapse of the affected lung, but the symptoms and dilatations remained as before.

Fig. *4* concerns a twenty-five-year-old male whose bronchiectasis was of ten years' duration. The pneumothorax in this case resulted in moderate improvement. The stippling indicates lipiodol in the alveoli. It is interesting to note the way normal alveolar tissue seems to surround the dilatations.

may result from this change of position. We have noted it in two cases.

Although pneumothorax treatment but seldom gives striking results in cases of bronchiectasis, it rarely does harm. By this is meant that the compression of the dilatations seldom results in such clinical manifestations of retention as fever and chills. In summarizing their experiences in cases of bronchiectasis treated by artificial pneumothorax, Whittemore and Balboni stated that in successful cases the pneumothorax must be kept up for a long time. In our experience this is frequently impossible unless the pneumothorax is replaced by an oleothorax. In true cases of bronchiectasis one should expect beneficial results early, and by true bronchiectasis we mean those cases in which the presence of the dilatations has been demonstrated by lipiodol. *If improvement does not take place within three to six months, then it is not likely to occur.* The expectoration is rarely completely abolished by a pneumothorax. In early cases of bronchiectasis one can some-

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Figs. *5* and *6*.—This patient received pneumothorax treatment for almost one year. The dilatations were compressed only slightly but the patient was relieved of her hemoptysis. She was subsequently subjected to a phrenicectomy and to oleothorax. Her case has been considered in detail in the discussion on oleothorax.

Fig. *7* refers to a male, aged sixteen years, who had right lower lobe bronchiectasis of eight years' duration (subsequent to a postoperative pneumonia). It was possible to obtain a very complete collapse (*P. C.*) in this case. In fact the collapse caused marked displacement of the trachea (*T.*), and mediastinal contents. *C. L.* indicates the collapsed lower lobe and *A.* an adhesion which contained lung tissue as demonstrated by lipiodol. This adhesion in no way prevented compression of the lower lobe. The degree of collapse far exceeded anything which could have been accomplished by any other form of compression therapy and yet everything but the dilatations were collapsed. In this case the pneumothorax was maintained for over a year but the patient failed to derive any benefit. A tonsillectomy was performed on November 28, 1930. Shortly after it was decided to abandon the pneumothorax and to undertake a thoracoplasty in preparation for a lobectomy. The eighth, ninth, tenth and eleventh ribs were removed on December 30, 1930 (E. A. G.). The patient had a rather stormy postoperative course and ran a very high fever. He developed a right-sided pleural effusion which never became infected. He began to improve gradually. This improvement was concomitant with the pulling over of the mediastinal contents to the right side and with the obliteration of the right pleural space. This patient was then discharged and advised to return later for a lobectomy. A communication from his physician, Dr. Johnson of Evansville, Indiana, stated that the patient had died on September 25, 1931.

Fig. *8* refers to a male, aged twenty-seven years, who suffers from bilateral bronchiectasis of fourteen years' duration. The condition apparently developed following a tonsillectomy which was performed under ether anesthesia. This patient was subjected to a pneumothorax, left, on March 17, 1930. This treatment was carried on until May 15, 1930. All his nasal sinuses were treated during this period and another tonsillectomy was performed on July 9, 1930. A right phrenicectomy was performed on May 17, 1930 (H. C. B.). The diaphragm failed to rise and the patient was not improved, in fact he became slightly dyspneic as a result of it. This patient has not benefited from the bilateral compression therapy or from the operations on his nasal sinuses.

Fig. *9* concerns a male, aged forty-eight years, who was admitted to Barnes Hospital on April 30, 1929, complaining of cough, foul yellowish, blood-streaked sputum, pain in the right chest, nervousness, loss of 8 pounds in weight and night sweats, all of over two years' duration. The onset was insiduous. In Fig. *9* one observes the original lesion, a chronic abscess of the lung. The dilatations at the base were not striking. The patient was placed on postural drainage and received neosalvarsan injections intravencusly. He was also subjected to pneumothorax treatment for many months with only slight relief. The basal bronchiectasis gradually became more prominent (October 14, 1929) and on November 19, 1929, the abscess cavity which had previously disappeared was again observed. A phrenicectomy was performed on January 24, 1930; the diaphragm rose appreciably. On January 28, 1930, the physical signs at the right base were typical of pneumonia. The patient rallied from this condition and then seemed to improve for a while. A lipiodol injection on October 17, 1930, revealed the large bronchiectatic abscess noted in Fig. 12. The patient was therefore subjected to a thoracoplasty on October 21, 1930, when portions of the seventh, eighth and ninth ribs were removed (E. A. G.). He did not do well after this operation and died on October 22, 1930. Necropsy by Dr. Pipkin revealed an abscess of the lung and bronchiectasis on the right side and an abscess of the lung in the region of the left hilus and an infected mural thrombus in the left ventricle. The abscess in the left lung had undoubtedly been present for some time. This case clearly indicates, among other things, that the lipiodol injection failed to show what was actually present. The dilatations were of the sclerotic variety and were filled with putty-like material. This material must have prevented adequate lipiodol injections at all times.

times do good by pneumothorax, but in the majority of chronic cases pneumothorax accomplishes but little, even if it is supplemented by a phrenicectomy. Pneumothorax treatment, however, does prepare the mediastinum for some future procedure, and it is perhaps indicated in the early forms of bronchiectasis in which the dilatations extend well to the periphery. This form of treatment may also be supplemented by postural drainage and the administration of neosalvarsan. What has been said concerning the pneumothorax treatment of bronchiectasis in the adult also holds for bronchiectasis during childhood. *Our results do not permit us to recommend pneumothorax in the treatment of bronchiectasis.*

PNEUMOTHORAX TREATMENT AS A PROPHYLACTIC MEASURE

The use of pneumothorax treatment in those inflammatory conditions of the lung which are known to predispose or result in the development of bronchiectasis has received but little attention up to the present time. It would seem that certain acute inflammatory conditions of the lung which are likely to become chronic and lead to the development of bronchiectasis might, with profit, be treated by pneumothorax. We are not recommending that all cases of acute lung suppuration or inflammation which are known to become chronic should be treated by pneumothorax.

BILATERAL COMPRESSION THERAPY FOR BILATERAL BRONCHIECTASIS

Bilateral compression therapy in cases of bilateral bronchiectasis has not been employed very often to our knowledge. Bilateral phrenicectomy has apparently been performed on a few occasions but there is no adequate available data as to its value. R. W. Matson reported two striking results. Bilateral pneumothorax in the treatment of bronchiectasis has received practically no consideration up to the present time. We have performed phrenicectomies on two patients who received compression therapy on the opposite sides. One of these patients was receiving pneumothorax treatment, which was later substituted by an oleothorax. A phrenicectomy was performed on the less diseased side during the period that he was receiving oleothorax treatment. The other patient was subjected to pneumothorax on one side and phrenicectomy on the other. This treatment was also supplemented by operations on all nasal sinuses and by postural drainage in one case. It is almost two years since this latter patient was treated, but he can hardly be considered to have improved as a result of the compression therapy. Both these patients complained of dyspnea following the phrenicectomies. This dyspnea did not disappear as is usually the case following phrenicectomy.

XIV. TREATMENT OF BRONCHIECTASIS BY OLEOTHORAX

That the injection of massive quantities of oil into the infected pleural cavity might result in its disinfection or that the oil might serve to cause compression in the case of pulmonary lesions has long been appreciated. Bernou realized this possibility clinically when he treated patients suffering from pulmonary tuberculosis who were receiving pneumothorax treatment, in this manner. We have used oleothorax in the treatment of bronchiectasis whenever we observed that the pneumothorax pocket was gradually becoming obliterated. It is possible to keep up compression for a much longer period by oleothorax than by pneumothorax. The latter must frequently be abandoned after six months because of adhesions. We are not in a position to state whether oleothorax will accomplish any more than

pneumothorax or other forms of compression. Certainly it does away with the necessity of frequent refills and permits one to keep up compression for a much longer period. Oleothorax is also said to help stabilize the mediastinum so that this form of treatment prepares the patient for a more radical procedure much as does pneumothorax. It may also be employed to obliterate a pleural space.

We employ the U. S. P. preparation of paraffin oil. To every 95 c.c. of liquid paraffin is added 5 c.c. of 5 per cent gomenol. For purposes of more exact roentgenologic visualization a few cubic centimeters of lipiodol or brominol may be added.

Two cases from our series are cited here. They illustrate some of the results to be expected.

CASE 9.—M. A. G., female, aged twenty-eight, first admitted to Barnes Hospital on November 13, 1928. Her complaints were hemoptysis, cough and mucopurulent expectoration. Her past history noted whooping cough at the age of five years, and considerable loss of time from school because of head and chest colds so that not unlikely the onset of the patient's condition dated back to childhood. Her tonsils and adenoids were removed when she was sixteen.

The physical examination including a lipiodol injection revealed left lower lobe bronchiectasis. The patient was therefore given pneumothorax treatment and at first made rather striking improvement. Her hemoptysis ceased. This pneumothorax treatment was continued at about ten-day intervals. It was supplemented by a phrenicectomy, left, which was performed April 25, 1929 (E. A. G.). The diaphragm rose but little following this procedure. The patient still continued receiving pneumothoraces in amounts varying up to 500 c.c., the final pressure always being slightly negative. The nasal sinuses were also treated because of the presence of a chronic sinusitis.

A satisfactory collapse of the left lung by pneumothorax treatment was obtained until January 2, 1930. Only 50 c.c. of air could then be injected before the pressure became positive. It was therefore decided to start oleothorax treatment (H. C. B.). Twenty c.c. of liquid paraffin were accordingly injected into the left pleural cavity on January 6, 1930, and 50 c.c. on the following day. Fluoroscopic examination at this time showed that the cavity was two-thirds filled. An additional 25 c.c. were injected on January 8, 1930. The cavity now contained 95 c.c. of liquid paraffin and was over three-quarters filled. On January 10, 1930, the fluid shadow seemed to be larger; this increase in the amount of the fluid content was undoubtedly due to pleural reaction. The pulse and temperature curves remained unchanged. Oil was withdrawn at this time and was cultured; no growth was obtained. Thirty c.c. of oil were injected on January 10, 1930, and 20 c.c. were injected on January 13 and January 17, 1930. Fifteen c.c. were injected on February 5, 1930. The total amount of oil injected was therefore 180 c.c. The pocket was completely filled.

The patient has not received any refills for over twenty months. She has gained in weight and her expectoration has been appreciably reduced since starting the oleothorax treatment. She has had one attack of hemoptysis during the past two years (Fig. 24 *H* and *G*).

CASE 10.—L. O., male, aged twenty-two years, had as his chief complaints cough and foul expectoration. They had been present since childhood. The expectoration amounted to several cupfuls per day. Physical examination including a lipiodol injection confirmed the diagnosis. The patient had marked clubbing of his fingers. He was found to be suffering from pansinusitis but would only submit to incision and drainage of the right maxillary sinus. This was carried out on February 25, 1930. Pneumothorax treatment, left, was started on March 17, 1930. The initial pressures were –2, 0. Twenty-five c.c. of air were injected; the final pressures were –1, +2. Seventy-five c.c. of air were injected on March 19; 25 c.c. on March

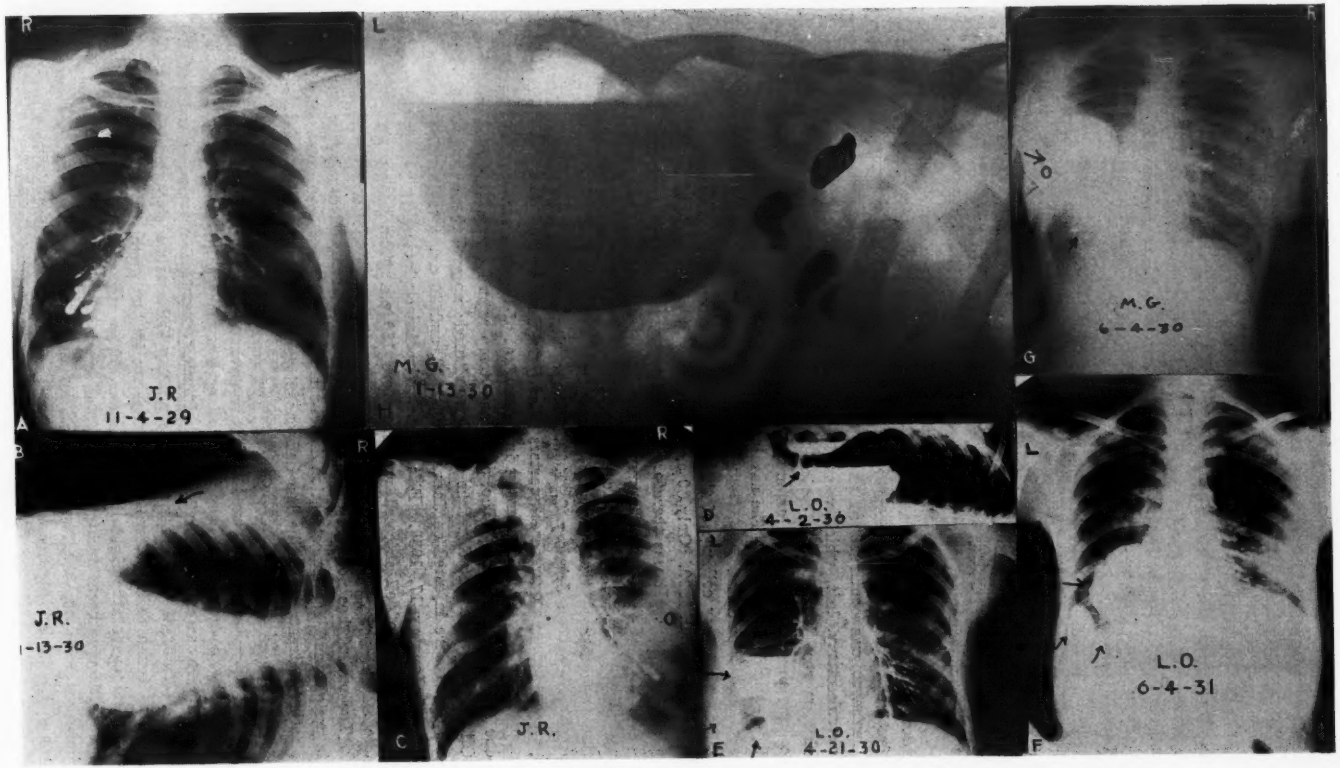
R
J.R
11-4-29
A
L
M.G.
1-13-30
R
M.G.
6-4-30
G
B
J.R.
1-13-30
R
C
J.R.
D
L.O.
4-2-30
L
E
L.O.
4-21-30
L
L.O.
6-4-31
F

Fig. 24

21; 100 c.c. on March 24 and 100 c.c. on March 28. The final pressures were then found to be −2, +4. The last pneumothorax refill was given on March 31, 1930, when the initial pressures were −8, −2 and the final pressures −2, +4. It was then decided to give the patient an oleothorax. This was done by Dr. H. M. Wilson. He introduced two needles into the pneumothorax pocket, obtained negative readings and injected 40 c.c. of liquid paraffin. The second oleothorax was given on April 7, 1930, when 60 c.c. of the same mixture were injected. Another 60 c.c. were injected on April 12, 1930, which made a total amount of 160 c.c. of liquid paraffin. It was found then that the oil had separated off into several pockets and that further injections were impossible. This oleothorax gave moderate collapse but improved the patient's cough and expectoration very slightly. A phrenicectomy, right, was performed (H. C. B.) on May 17, 1930. The diaphragm rose but little. Following this procedure the patient developed some dyspnea which partly disappeared gradually. In this case the combined treatment was ineffective. During October, 1931, this patient died at his home. The limitations of collapse therapy in this case are obvious (Fig. 24 *D*, *E* and *F*).

XV. INTRATHORACIC COMPRESSION OTHER THAN PNEUMOTHORAX IN THE TREATMENT OF BRONCHIECTASIS

Graham in 1921 was one of the first to recommend intrathoracic compression by gauze packs in the treatment of bronchiectasis. Zaaijer in 1927 reported a case in which preliminary phrenicectomy was followed by rib resection which included the periosteum and intercostal muscles. The diseased lobe was then isolated intrathoracically and was tamponed. The tamponed isolated lobe was finally incised and resected. This procedure was apparently quite successful. Compression of this sort at first glance seems to recommend itself, particularly because it does not call for the removal of much of the chest wall. It is only necessary to remove a small segment of one or more ribs. Whether the compression is by a pack or paraffin is perhaps not of great moment. It would seem that in cases in which pneumothorax is unsuccessful a preliminary period of compression by a paraffin plombe might prepare the mediastinum for later thoracoplasty, and eventually for lobectomy if necessary. Schrötter reported a case of bronchiectasis which was successfully treated by Sauerbruch in 1928 by phrenicectomy and extrapleural plombage. Rogers recently stated that Sauerbruch has used plombe most extensively in the surgical treatment of lung abscess and bronchiectasis. We have been unable to find any published report which deals with the results in these cases. Plombe has apparently been employed in bilateral cases. Wiese has recently reported experiences in ten cases in which the operation was performed on children suffering from bronchiectasis. He used the following preparation:

Paraffin (melting point 52° C.)	150.0
Paraffin (melting point 43° C.)	50.0
Bismuth Carb. neutr.	2.0
Vioform	0.1

Fig. 24.—Oleothorax in the treatment of bronchiectasis.

A. (J. R.) The bronchiectasis in this case resulted from an attack of influenza in 1918. This patient was first subjected to pneumothorax treatment and then to oleothorax. Fig. *B* would suggest that striking compression had been obtained but film *C* indicates that the dilatations are not compressed at all. (O = oleothorax.) This patient's complaints were moderately improved at the start.

Figs. *D*, *E* and *F* concern the patient referred to in Case 10 in the text. He received bilateral compression therapy for his bilateral lesion. Note the fluid levels in film *D*, which was taken with the affected side uppermost. The arrows indicate the numerous pockets into which the oil collected.

Figs. *H* and *G* refer to Case 9 of the text. The nature of the compression and the distribution of the dilatations (retouched) are shown in Fig. *H*. These dilatations ride over the diaphragm and are not compressed by the oleothorax. The roentgenologic appearance of this patient's chest at the present time corresponds in every respect with Fig. *G*. She has not received any refills for over twenty months and is definitely improved.

Four of the patients so treated were females; six were males. One was seven years of age, two between eight and nine, three between ten and eleven, four between twelve and sixteen. The left side was treated on nine occasions, the right once. Eight of the operations were performed under local anesthesia. The largest amount of paraffin mass employed was 530 grams, the least 200 and the average about 400 to 450 grams. In two cases pneumothorax and tearing of the lung with subcutaneous emphysema resulted, but the operation was continued. In one case asphyxia resulted because of too strenuous compression and on another occasion postoperative dyspnea because of too tight bandage. There were no deaths and the immediate results were striking in over half the cases. Since most of these operations were performed less than two years ago, it is obviously too soon to attempt to draw any conclusions.

Compression of Bronchiectatic Dilatations by Specially Constructed Belt, Applied Following Partial Thoracoplasty.

CASE 11.—B. A., female, aged thirty-eight. First admitted to Barnes Hospital in 1921, complaining of cough, foul expectoration, occasionally blood streaked (amount up to 13 quarts per day, 200 to 300 c.c. of blood at times), loss of weight. The onset of the illness was apparently in 1917, following influenza. The patient went to the country and to a sanatorium and received 15 pneumothorax treatments (right). Her sputum was free from acidfast bacilli, but contained numerous spirochetes. Bronchoscopy was undertaken on January 26, 1921, but proved unsuccessful. Five inches of the seventh and eighth ribs, right, were resected on January 31, 1921 (E. A. G.). The pleura was opened. The lung was found firmly adherent to the parietal pleura and required sharp dissection to release it. The right lower lobe was separated everywhere from the chest wall, diaphragm and pericardium. By suturing the parietal pleura and reenforcing it by suturing of the diaphragm to the chest wall an effort was made to close the lower lobe in a constricting pocket in order to secure compression. Closure was made without drainage and the patient was given a specially constructed belt to maintain this compression. She gave birth to a normal child in 1926, but an examination of her chest in that year showed no appreciable improvement of the pulmonary signs and symptoms. She was last observed in December, 1930. Her complaints were still cough, mucopurulent expectoration, occasionally blood streaked, and weakness. Physical signs were still as before. Several lipiodol injections were successful. Patient was given a course of neosalvarsan. Because of her age (she is now forty-seven) and the fact that she is the mother of several small children it was decided not to consider any more radical treatment which would otherwise be indicated.

XVI. TREATMENT OF BRONCHIECTASIS BY THORACOPLASTY

By uncomplicated bronchiectasis Hedblom means the characteristic changes of that disease in a portion of the bronchial tree without parenchymatous abscess formation as demonstrated by the roentgenogram. By graded extrapleural thoracoplasty he implies a collapse of the wall of the chest, that is, paravertebral resection of the upper eleven ribs in several operative stages. In order to secure complete collapse of the base without collapsing the normal upper portion of the lung, he states that a complete resection of the whole length of the lower ribs is necessary and that resection of the third and fourth ribs must often be included in order to prevent their suspending effect on the lower part of the chest wall. Concerning the seven patients upon whom he performed a graded extrapleural thoracoplasty without mortality in 1923, he stated in 1927 that the boy, aged nineteen years (Case 1), whose sputum previously averaged 240 c.c. to 500 c.c. in twenty-four hours and whose third to eleventh ribs were resected in five stages, was practically free from symptoms but

continued to raise 60 c.c. to 150 c.c. of sputum in twenty-four hours. A man, twenty-eight years of age (Case 2), who previously raised 500 c.c. to 1000 c.c. of sputum in twenty-four hours, now had an average sputum of 60 c.c. in twenty-four hours during the six years which had elapsed since the operation. A female, aged twelve years (Case 3), who had a postinfluenzal bronchiectasis, had gained in weight (forty pounds) and the expectoration had been reduced from 500 c.c. to 30 c.c. Case 4 failed to show any striking improvement—this was a posttonsillectomy bronchiectasis. Case 5, a boy, aged 14 years, nine months after the operation suffered from abscesses which formed in the soft tissue of the chest wall. The condition was then found to be actinomycosis. The patient died of general asthenia a month later. Case 6 concerned a girl, aged eighteen years, with a bronchiectasis of nine years' duration of the postpneumonic type; this patient was subjected not only to partial thoracoplasty but to a partial cautery lobectomy. Her sputum has been reduced from 480 c.c. to 60 c.c. A female, aged twenty-two years (Case 7), with chief complaints of hemoptysis and purulent expectoration (about 240 c.c.), gained weight and had her expectoration reduced to about 30 c.c. to 60 c.c. following thoracoplasty.

The most recent report published by Hedblom which deals with his results in the treatment of bronchiectasis by thoracoplasty appeared in the early part of 1931. Hedblom then stated the following: "In 1926 I reported 14 cases in which I had done a thoracoplasty in five stages without operative mortality. Three of the patients were entirely relieved and have remained symptom-free since. Seven of the patients have continued to raise sputum amounting to 10 per cent to 20 per cent of what had been raised before the operation. Six of the seven have been working regularly. Four, whose symptoms were greatly relieved, died from ten months to two years after operation, one from actinomycosis infection, one from drug poisoning, one from pulmonary hemorrhage, and one following secondary operation."

Hedblom reports that 18 additional patients have been operated upon since, several by a three stage operation. Four of these patients died, one on the ninth day following a first stage resection of four ribs. This patient died from brain abscess. The second patient died on the seventh day after a similar first stage operation from a retention pneumonia, the third on the eighth day after a second stage operation from pulmonary gangrene and the fourth from symptoms suggesting pneumonia. The fourteen remaining patients were all improved in greater or lesser measure, the sputum being reduced from 60 per cent to 90 per cent. Two of these patients later died after secondary lobectomy and one from pulmonary hemorrhage. Of the thirty-two patients subjected to thoracoplasty by Hedblom, twenty-one are still alive.

Hedblom believes that thoracoplasty produces a degree of collapse which can be made to approximate that following artificial pneumothorax. He believes that the most favorable type is the early peripheral cylindrical lesion in patients in whom phrenic evulsion has not produced sufficient improvement. It is his opinion that early thoracoplasty will relieve the excessive grade of negative intrapleural tension incident to an atelectasis and that it will so prevent the formation of sacculations. It is also Hedblom's opinion that bronchiectasis associated with multiple abscess formation clinically manifested by septic temperature, leucocytosis, etc., is not the type of bronchiectasis to be treated by thoracoplasty.

Hedblom in 1924 summarized some of the opinions expressed in the literature with regard to results of thoracoplasty for nontuberculous pulmonary suppuration. He stated that Hansen tried the method in two cases without benefit and that Mumford

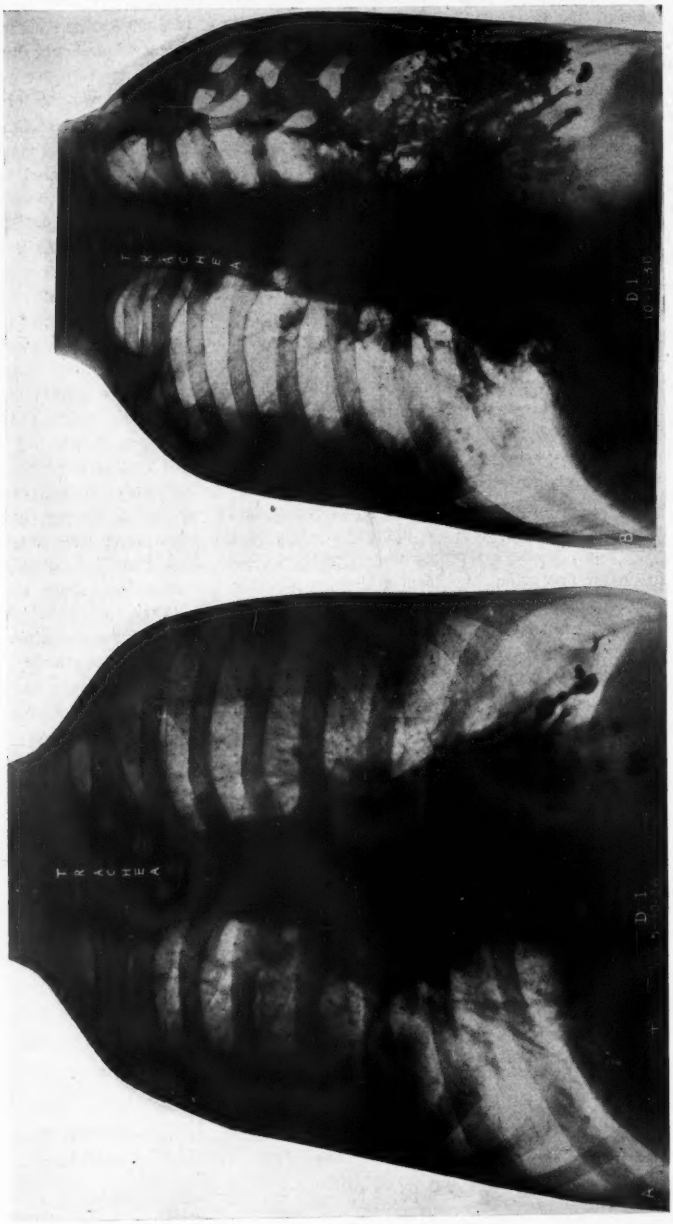

Fig. 25

and Robertson lost two patients and nearly lost a third while three of Lilienthal's patients improved and one died. Lilienthal has reported an instance in which a striking result had been obtained by thoracoplasty. Harrington's early experiences concerned 17 cases in which there was one death from bronchiectatic abscesses. His results at the time of reporting were only of two years' duration. He usually performed a surgical collapse in three or four stages. In a recent publication (December, 1931) on the surgical treatment of bronchiectasis and lung abscess Harrington reported the extrapleural resection of ribs in 31 cases. It is not quite clear as to whether all these cases were cases of bronchiectasis. There were 3 operative deaths. Eight patients were improved to the extent of 75 to 90 per cent relief of symptoms. Zinn and Muhsam and Küttner have reported single cases in which improvement followed the operation. Davies also treated a patient in this manner and DeQuervain performed a lobectomy in one case following thoracoplasty. Lenhartz reported one cure and one improvement, and Körte one "cure," and one uncertain result. Sauerbruch (cited by Denk) has reported 49 cases. There were 2 operative and 6 late deaths in the series. Seventeen patients were improved. Brauer (cited by Denk) in 1925 reported 8 cases with rather satisfactory results. There were no cures. Denk has reported 6 cases with poor results.

Thus, in this review of the literature one is impressed with the fact that the results obtained have not been altogether striking. One can also note, however, that according to the literature relatively few patients suffering from bronchiectasis have been subjected to thoracoplasty. Certainly some of the poor results could be attributed to the fact that the operation was not divided into a sufficient number of stages, also that in certain cases more than uncomplicated bronchiectasis was being treated. It would seem that lack of stabilization of the mediastinum is frequently the cause of the poor results and that the removal of ribs in certain cases of bronchiectasis seems to deprive the mediastinum of the necessary support. Although the results obtained in the treatment of bronchiectasis by thoracoplasty leave much to be desired, certainly this procedure serves as a preliminary to other procedures which are best undertaken following the thoracoplasty. What Hedblom in 1924 stated is, therefore, true, namely, that if the results are unsatisfactory lobectomy can then be performed. One must always be prepared to expect a failure to obtain satisfactory compression of the dilatations no matter how extensive the thoracoplasty has been.

Fig. 25.—Extrapleural thoracoplasty (Sauerbruch) for bronchiectasis, left.
(E. D. I.) A male, aged twenty-four, was admitted to Barnes Hospital May 4, 1926. His complaints were cough, expectoration and dyspnea of many years' duration. His past history made mention of a rib resection for an empyema, left, when he was eighteen years of age. At the age of twenty-two, he was treated for a lung abscess. This operation was done in two stages. The first operation was interrupted because of collapse of the lung. This fact suggests that the previous resection was not done for the drainage of an empyema. The lung was sutured to the pleura but a subsequent attempt to locate the abscess was unsuccessful. He was admitted to Barnes Hospital on May 7, 1926. A lipiodol injection revealed the presence of cylindrical bronchiectasis in the left lower lobe. Pneumothorax was attempted on May 14, 1926, but proved unsuccessful because of adhesions. The first stage of a thoracoplasty was performed (E. A. G.) on May 18, 1926. The operation was completed on June 3, 1926. The patient's cough and expectoration were only slightly improved by the thoracoplasty. Although the degree of collapse is not great, and although film *B* was not taken under quite the same condition as *A*, still it indicates quite clearly that the collapse obtained was sufficient to displace the trachea. The patient was observed again during October of 1930. A lipiodol injection performed on October 1, 1930, showed that the dilatations were still present. A phrenicectomy was performed on October 8, 1930. It was followed by a slight rise of the diaphragm but no clinical improvement. This patient will probably be subjected to a lobectomy at a future date.

Fig. 26.—Thoracoplasty in the treatment of bronchiectasis.
The amount of collapse obtained in this case was sufficient to displace the trachea yet the dilatations (outlined by lipiodol and indicated by arrows) still persisted. (S = stomach, which contains some swallowed lipiodol.)

Our own results following thoracoplasty for bronchiectasis have not been striking. (Figs. 25 and 26.)

XVII. PHRENICECTOMY IN THE TREATMENT OF BRONCHIECTASIS*

Little or no attempt has been made to determine whether the results obtained in the treatment of bronchiectasis by phrenicectomy are permanent. This is perhaps due to the fact that only since the introduction of lipiodol have we been able to make an accurate diagnosis of bronchiectasis in most instances. Phrenicectomy in the treatment of bronchiectasis is therefore of only recent development, but in spite of this fact this simple operation is being performed freely. We have therefore deemed it advisable to consider this form of treatment at length. There are surprisingly few recorded cases of bronchiectasis in which phrenicectomy has been the sole form of treatment. Is the reason for this the limitations of the method or that the good results obtained are not lasting? The beneficial effects are considered to be due to the production of more adequate drainage. We are of the opinion that in some cases the bronchi become more horizontal and that this results in more adequate drainage. (See Fig. 23, *1* and *2*.) There are some who believe that the dilated bronchi become compressed. One certainly doubts whether any significant collapse of true bronchial dilatations is ever accomplished by phrenicectomy alone. The symptomatic beneficial effects have been said to be striking and permanent by many. An attempt has been made to determine the value of phrenicectomy as a therapeutic measure in 82 cases. This number is made up of 26 cases from the Barnes Hospital and 56 cases selected from the literature. These observations were made early in 1931. Undoubtedly many more cases are available now.

Effect of Phrenicectomy Upon Cough.—

Without going into the mechanism of cough and whether or not phrenicectomy should theoretically affect it, one can say that there are few occasions in which cough is completely abolished by this procedure. Phrenicectomy rarely aggravates the cough. Of greater significance is the fact that the abolition of cough may cause harm, by that is meant that expectoration is made more difficult and sometimes almost impossible. Wilson's experiences in this connection coincide closely with our own. He stated that the expectoration in 18 patients with bronchiectasis was easier after phrenicectomy in four, unchanged in eleven, and more difficult in three. It is, of course, appreciated that the patient's cough may be influenced but the expectoration not at all. The converse is likewise true.

Expectoration.—

Phrenicectomy may reduce the amount of expectoration to a striking extent. It may even cause its disappearance. This effect is unfortunately not always lasting.

*Much of the labor of compilation of cases from the literature on the effect of phrenicectomy on bronchiectasis has been performed by David Betsui to whom we wish to express our thanks.

Fetor.—

The effect of phrenicectomy upon fetor has been well illustrated by Wilson. This is noted in the table from his work (Table IV).

We have never observed any striking effect upon fetor in cases of bronchiectasis treated only by phrenicectomy.

TABLE IV

Number	Age	Duration of disease	*Sputum.* Quantity per day c.c.	*Sputum.* Fetor	*Artificial Pneumothorax Therapy Effect on Sputum* Duration mos.	*Artificial Pneumothorax Therapy Effect on Sputum* Quantity c.c.	*Artificial Pneumothorax Therapy Effect on Sputum* Fetor	Period since phrenic evulsion mos.	*Effect of Combined Artificial Pneumothorax and phrenic evulsion on sputum* Quantity c.c.	*Effect of Combined Artificial Pneumothorax and phrenic evulsion on sputum* Fetor
188	35	3	226	–	2½	226	–	2	226	–
2	38	1½	?	?	15	142	+	12	142	+
162	30	12	60-80	+	1½	60-80	+	5	14-28	–
169	17	a	114	+	15	57	+	4	tr.	–
25	26	12	85	+	3	57	+	36	0	–
22	21	2	114	+	1½	114	+	5	57	+
167	26	10	250	+	4	28	⊕	3	28	–
48	10	1	114	+	3	57	+	18	tr	–
77	15	7	tr.	+	6	0	–	21	0	–
116	19	3	220	+	4	226	+	12	226	+
76	32	¾	284	+	¾	284	+	21	14	⊕
80	19	4	170	?	x			18	142	?
63	15	7	60-110	±	24	0	–	24	60-110	⊕
8	22	1	170-250		6	170-250		48	85-140	
195	20	b	500-700	+	6	284	+	6	114	±

a — since early childhood.
b — since infancy.
– — not offensive.
+ — offensive.
± — sometimes offensive.
⊕ — less offensive.
x — phrenic evulsion three months before artificial pneumothorax.

*Wilson, Harold: Med. J. Australia **2**: 487, October 11, 1930.

Hemoptysis.—

It was hoped that since bronchiectasis is so frequently basal, phrenicectomy would be a valuable means of treating hemoptysis. It is as yet impossible to draw any definite conclusions in this connection since sufficient cases are not available. We have observed one striking result. Others have reported similar striking results.

Dyspnea.—

Dyspnea in cases of bronchiectasis is a most important preoperative consideration. It constitutes a definite contraindication to phrenicectomy in certain cases. If the dyspnea is due to adhesions between the pericardium and diaphragm then phrenicectomy may be helpful. One should rather conclude, however, that the phrenicectomy will increase the degree of dyspnea. Strangely enough, this dyspnea does not always prevent the patient from being moderately active. And so it is an error to conclude, since the patient only states that he is dyspneic on exertion and yet able to walk, that the dyspnea is not of serious moment. The electrocardiogram may fail to show changes other than what might be considered due to alteration of the cardiac axis. Even the vital capacity may show but slight change. But this slight change

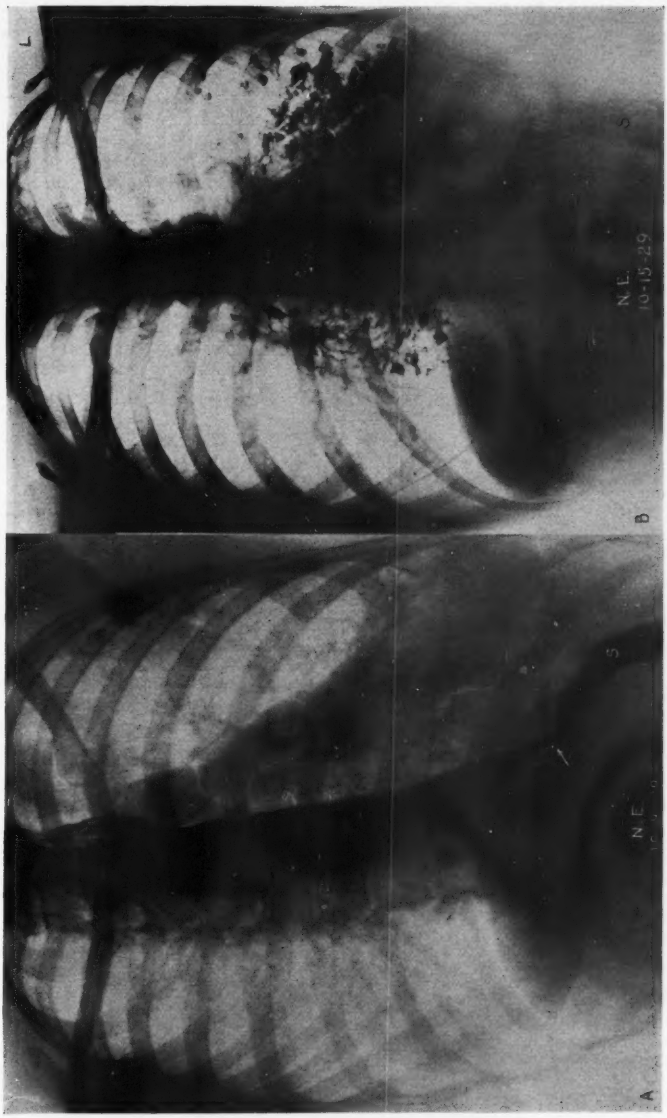

Fig. 27.—Cardiospasm following left phrenicectomy for bronchiectasis. (S = stomach.)

The patient in this case, a female, aged thirty-six, with bilateral bronchiectasis, was subjected to a left phrenicectomy on April 12, 1928. She developed dysphagia two months later. The film (*A*) made during October, 1929, shows an even dilatation of the esophagus typical of "cardiospasm."

Fig. *B*, obtained two days after an esophagoscopic examination, shows the esophagus outlined with barium and the bilateral dilatations outlined with lipiodol. The left diaphragm is appreciably elevated. There is no marked angulation of the abdominal portion of the esophagus. This patient's dysphagia improved following the administration of atropine sulphate.

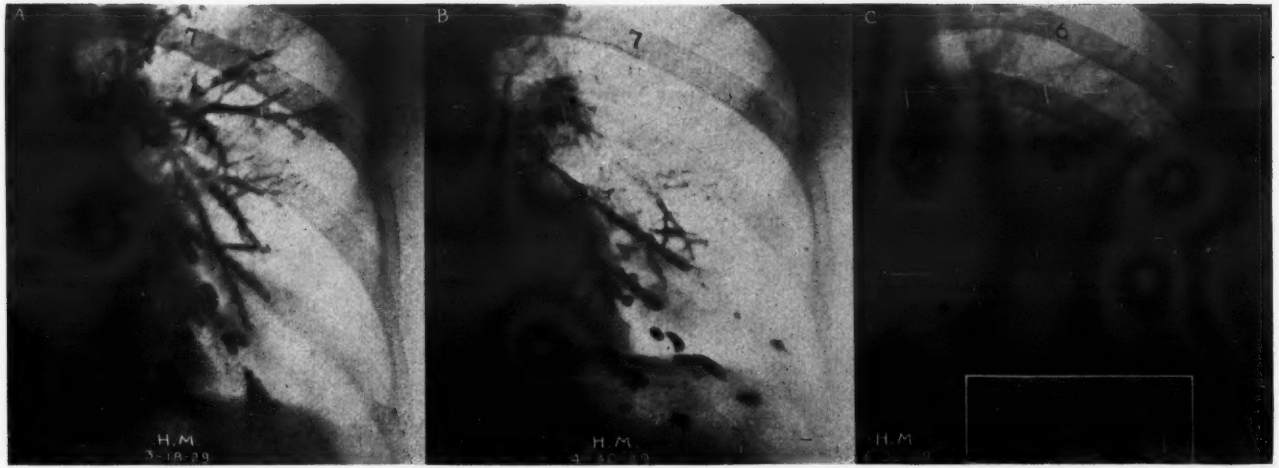

Fig. 28.—Phrenicectomy in the treatment of bronchiectasis.

H. M., male, aged fifty-seven, was admitted to Barnes Hospital on March 18, 1928, complaining of cough, weakness and fever of about ten years' duration. He was found to be suffering from left lower lobe bronchiectasis (*A*, March 18, 1929). A phrenicectomy was performed on March 23, 1929. The patient's condition did not improve following it. Shortly after he began to experience difficulty in raising his sputum. Because of definite evidence of chronic maxillary sinusitis Doctor Arbuckle performed a Caldwell-Luc operation (left) on April 2, 1929. He found the antrum lined with pus and thick pyogenic membrane. The operation was repeated on the right side on April 16, 1929, and the ethmoid and sphenoid sinuses were then incised and drained. The antral mucous membrane was very hyperplastic but not polypoid. There was no pus. This patient was definitely sensitive to lettuce, chicken, pork, duck and wool, and so his diet was controlled, but without benefit.

Fig. *B*, taken on April 30, 1929, shows that the course of the bronchi has apparently been altered by the paralyzed and slightly elevated diaphragm.

Fig. *C*, taken on June 22, 1929, shows that the dilatations (outlined) still contained some of the lipiodol (indicated in rectangle), which was injected on April 30, 1929. Note also the extensive pneumonic process and lung abscess. This patient died on June 23, 1929.

may be sufficient to produce dyspnea since a significant reduction of vital capacity has in most cases already occurred as a result of disease.

Does Failure of the Diaphragm to Rise Following Phrenicectomy Influence the End-Results?

It would seem that beneficial results of phrenicectomy in cases of bronchiectasis are nearly always observed if the diaphragm rises. In a large percentage of cases in which the results were poor the diaphragm failed to become elevated following its paralysis.

Effect of Phrenicectomy Upon the Physical Signs, Size of the Dilatations and Progress of the Disease.—

The physical signs are usually uninfluenced by the phrenicectomy, even in cases in which the benefits are striking. The size and shape of the dilatations remain unaltered. Of great significance is the fact that it is possible for the cough and expectoration to be definitely improved by the phrenicectomy and yet for the disease to progress. It has been said that the age of the patient and the duration of the disease do not influence the result. We cannot entirely agree with this statement inasmuch as in cases of atelectatic bronchiectasis and with much mediastinal displacement the beneficial effects are always less striking. Such types of bronchiectasis are undoubtedly of long standing. We have observed the most striking effects in cases in which dilatations were not marked, the history of illness of rather short duration and the degree of peribronchial fibrosis slight. The distribution of the bronchiectasis is also of some import, likewise whether the condition is unilobar, multilobar, unilateral or bilateral. Perhaps those cases in which the bronchitis is the primary cause do better than those in which the bronchiectasis is secondary to chronic suppuration or abscess formation. Phrenicectomy alone rarely suffices in either case. Sometimes it seems as if the associated parenchymal disease is influenced much more than the dilatations and as if the benefits derived are due to the effects of the phrenicectomy upon it.

Ill Effects of Phrenicectomy in Cases of Bronchiectasis.—

Reference to some of the effects of phrenicectomy upon the heart, esophagus and stomach have been made elsewhere. We need here only note that following left phrenicectomy cardiospasm apparently developed in one patient with bilateral bronchiectasis (Fig. 27). Another case of cardiospasm following right-sided phrenicectomy has recently been reported by Blask. The effect on dyspnea has also been considered. Phrenicectomy may so hinder expectoration as to cause serious retention of secretions (Fig. 28).

The early results may be striking and may, therefore, be misleading. They may be misleading because the reduction in cough and expectoration may erroneously suggest that the disease is not progressing. The ideal time for a more radical, more satisfactory procedure, one with more possibilities may be lost. The opposite lung may become involved during the period of so-called well being and if the original focus had been treated this manifestation in the opposite lung might have been averted. We have recently had one striking example of this fact.

The 82 cases of bronchiectasis in which phrenicectomy was performed are tabulated in Tables V and VI. By "definitely improved" we mean

TABLE V

BRONCHIECTASIS TREATED BY PHRENICECTOMY

Cases from Literature

Total Number of Cases—56

D. I. = Definite Improvement. M. I. = Moderate Improvement. S. I. = Slight Improvement. N. I. = No Improvement. U. = Uncertain. W. = Worse.

RESULTS DUE TO PHRENICECTOMY ALONE	NO.*	ETIOLOGY	UNILATERAL INVOLVEMENT	BILATERAL INVOLVEMENT	RESULTS OF AUXILIARY TREATMENTS			
					BEFORE PHRENICECTOMY	RESULT	AFTER PHRENICECTOMY	RESULT
D. I.	1		+				Postural drainage	
	2	Postpneumonic	+		Costal resection Pneumothorax Partial pneumolysis	N. I. N. I. N. I.		
	3	Postpneumonic	+		Postural drainage	N. I.		
	4	Gunshot wound '18	+					
	5	Postinfluenzal pneumonia	+					
	6	Extension from other lung		+ later	Pneumothorax, lt. Pneumothorax, rt.			
(19)	13	Cough and sputum since 1910 Exacerbation, 1916 post pneumonic	+					
	15		+					
	18		+					
	19	Postpneumonic	+					
	22	Exacerbation 1921 "cold"	+					
	38		+					
	39		+					
	48		+				Thoracoplasty	

*The numbers in this column denote references in the Bibliography under "Phrenicectomy."

TABLE V (CONTINUED)

RESULTS DUE TO PHRENICECTOMY ALONE	NO.*	ETIOLOGY	UNILATERAL INVOLVEMENT	BILATERAL INVOLVEMENT	RESULTS OF AUXILIARY TREATMENTS			
					BEFORE PHRENICECTOMY	RESULT	AFTER PHRENICECTOMY	RESULT
	49		+					
	50			+				
	51			+				
	52		+					
	53		+					
M. I.	27		+				Antisyphilitic	
(4)	9		+		Vaccine therapy Pneumothorax	N. I.		
	23		+		Pneumothorax	D. I.	Postural drainage	D. I.
	35	"Cold" 9 mo. prev.	+		Med. treatment			
S. I.	29	Had bronchiectasis 6 yr.	+					
	20	Onset at 5 yr. Exacerbation at 34 due to flu	+		Med. treatment	D. I.		
(5)	25			+ mainly on right			Postural drainage	
	40		+				Thoracoplasty	
	55		+					
N. I.	8		+		Pneumothorax			

TABLE V (CONTINUED)

RESULTS DUE TO PHRENICECTOMY ALONE	NO.*	ETIOLOGY	UNILATERAL INVOLVEMENT	BILATERAL INVOLVEMENT	RESULTS OF AUXILIARY TREATMENTS			
					BEFORE PHRENICECTOMY	RESULT	AFTER PHRENICECTOMY	RESULT
	11	Unknown. 3 yr. duration	+		Pneumothorax Thoracoplasty	N. I. N. I.	Thoracoplasty Lobectomy	N. I. D. I.
	12		+		Pneumothorax		Rib resection drainage of pul. abscess	W.
	14		+					
	17	Since childhood	+		Med. treatment		Ligation of pul. artery	W.
	16		+		Pneumothorax			
	21		+					
(18)	24		+				Thoracoplasty Cautery pneumectomy	S. I.
	26			+ mainly on right			Thoracoplasty Appendectomy	W.
	30	Postpneumonic	+		Paravertebral thoracoplasty			
	32			+ mainly on right			Postural drainage	
	33		+				4 stage lobectomy	D. I.
	34		+				Thoracoplasty	W.
	37		+		Pneumothorax	N. I.	Partial thoracoplasty	W.
	43		+		Thoracoplasty	No data		

TABLE V (CONTINUED)

RESULTS DUE TO PHRENICECTOMY ALONE	NO.*	ETIOLOGY	UNILATERAL INVOLVEMENT	BILATERAL INVOLVEMENT	RESULTS OF AUXILIARY TREATMENTS			
					BEFORE PHRENICECTOMY	RESULT	AFTER PHRENICECTOMY	RESULT
	44		+		Thoracoplasty	No data		
	45		+					
	47		+					
W.	10		+		Vaccine therapy Pneumothorax	N. I.	Pneumothorax	N. I.
(6)	28	Postpneumonic and postempyemic	+					
	31		+				Postural drainage	
	36	8 yr. duration	+		Bronchoscopy for removal of f. b.	M. I. temp.		
	46		+					
	54		+					
U.	7		+		Thoracoplasty			
	41		+		Thoracoplasty	No data		
(4)	42		+				Thoracoplasty	
	56	Postpneumonic	+		Med. treatment Pneumothorax	N. I. N. I.	Lobectomy	D. I.

TABLE VI

Bronchiectasis Treated by Phrenicectomy

Barnes Hospital Series

Total Number of Cases—26

D. I. = Definite Improvement. M. I. = Moderate Improvement. S. I. = Slight Improvement. N. I. = No Improvement. U. = Uncertain. W. = Worse.

RESULTS DUE TO PHRENICECTOMY ALONE	ETIOLOGY	UNILATERAL INVOLVEMENT	BILATERAL INVOLVEMENT	RESULTS OF AUXILIARY TREATMENTS			
				BEFORE PHRENICECTOMY	RESULT	AFTER PHRENICECTOMY	RESULT
D. I.	Cough and sputum for 20 years	+		Incision and drainage of maxillary sinus			
(3)	"Flu" 1924	+		Pneumothorax	N. I.		
	"Cold" 18 yr. ago, flu 1921, exacerbation	+		Pneumothorax Bronchoscopy	D. I.		
M. I.	Postoperative pneumonia 1925	+		Lipiodol injection and bronchoscopy for diagnosis			
	"Cold spells" Feb. '27		+	Pneumothorax	M. I.	Postural drainage	?
(5)	"Cold" 2 yr. ago, 1924	+		Sulpharsphenamine Thoracoplasty Cautery pneumectomy	D. I. D. I.		
	"Flu" 10 yr. ago	+		Postural drainage Rest pneumothorax	 D. I.		
	"Cold" 10 yr. ago	+	More marked on rt.	Bronchoscopy, X-ray Neoarsphenamine and Lugol's sol. Pneumothorax	 D. I. D. I.	Incision and drainage of maxillary sinuses (rt. and lt.)	D. I.
S. I.	Chr. cough at 5 yr. of age		+	Incision and drainage of ethmoidal and sphenoidal and maxillary sinuses. Pneumothorax	 S. I.		
(4)	"Flu" 1918	+		Pneumothorax	N. I.	Op. for mastoiditis	D. I.

TABLE VI (CONTINUED)

RESULTS DUE TO PHRENICECTOMY ALONE	ETIOLOGY	UNILATERAL INVOLVEMENT	BILATERAL INVOLVEMENT	RESULTS OF AUXILIARY TREATMENTS			
				BEFORE PHRENICECTOMY	RESULT	AFTER PHRENICECTOMY	RESULT
	Scarlet fever at 2 yr.		+	Bronchoscopy Pneumothorax	M. I. N. I.	Neosalvarsan	S. I.
	Upper respiratory infection	+		Pneumothorax	D. I.		
N. I.	Cough all through life Exacerbation 2-3 yr. ago	+				Pneumothorax Op. on nasal sinuses	D. I.
	Postpneumonic 17 yr. ago	+		Sinus operation Bronchoscopy	N. I.		
	Foreign body (gold band tooth) aspirated during anesthesia for appendectomy, 1921		+	Thoracostomy Bronchoscopy Pneumothorax	M. I. N. I.	Sinus operation	S. I.
(6)	Cough and sputum 12 yr.		+	Pneumothorax Sinus operation	N. I. S. I.	Tonsillectomy and adenoidectomy Maxillary (rt. lt.) sphenoidal (rt. lt.) and ethmoidal sinuses incised and drained	S. I.
	Chr. bronchitis all through life	+		Maxillary antrum incision Pneumothorax	 N. I.		
	Postpneumonic	+		Salvarsan Thoracoplasty		Thoracoplasty	D.
U.		+		Pneumothorax Thoracoplasty	N. I. S. I.		
(3)	Bronchiectasis at 10 yr. of age	+		Pneumothorax Postural drainage	D. I. D. I.	Cautery lobectomy	D. I.

TABLE VI (CONTINUED)

RESULTS DUE TO PHRENICECTOMY ALONE	ETIOLOGY	UNILATERAL INVOLVEMENT	BILATERAL INVOLVEMENT	RESULTS OF AUXILIARY TREATMENTS			
				BEFORE PHRENICECTOMY	RESULT	AFTER PHRENICECTOMY	RESULT
		+		Bronchoscopy. Thoracoplasty. Cautery pneumectomy. Thoracostomy Pneumothorax	N. I.	Rib resection	S. I.
W. (2)	Bronchitis, 22 yr. ago	+					N. I.
	2 yr. ago cause not mentioned	+		Pneumothorax	S. I.	Thoracoplasty	W.
Op. done to reduce size of anatomical defect (3)	"Grippe" 1928 resulted in flare-up	+		Bronchoscopy Pneumothorax Pneumectomy, cautery	D. I. N. I. D. I.		
	Postpneumonic	+		Thoracostomy Pneumectomy Adenoidectomy	M. I. D. I.		
	Foreign body 17 yr. ago	+		Pneumothorax Pneumectomy Extraction of teeth	N. I. D. I.	Partial resection of wall. Op. for fistula and thoracoplasty	D. I.

TABLE VII—A

BRONCHIECTASIS TREATED BY PHRENICECTOMY

Barnes Hospital Series

	NO. OF CASES	RESULTS
Late results attributable to phrenicectomy and not to other pre- or postoperative measures	3	Definitely Improved
	5	Moderately Improved
	4	Slightly Improved
	6	Not Improved
	3	Uncertain
	2 ?	Worse
Operations done to reduce size of anatomical defects	3	Moderately Improved
Total	26	

TABLE VII—B

BRONCHIECTASIS TREATED BY PHRENICECTOMY

Cases From Literature

	NO. OF CASES	RESULTS
Late results attributable to phrenicectomy and not to other pre- or postoperative measures	19	Definitely Improved
	4	Moderately Improved
	5	Slightly Improved
	18	Not Improved
	6	Worse
	4	Uncertain
Total	56	

TABLE VII—C

BRONCHIECTASIS TREATED BY PHRENICECTOMY

Cases From Literature and Barnes Hospital

	NO. OF CASES	RESULTS
Late results attributable to phrenicectomy and not to other pre- or postoperative measures	22	Definitely Improved
	9	Moderately Improved
	9	Slightly Improved
	24	Not Improved
	8	Worse
	7	Uncertain
Phrenicectomy done to reduce size of anatomical defects	3	
Total	82	

that the complaints were completely relieved. This does not imply that the physical signs or the lesion disappeared. By "moderately improved" we mean that even though the signs and symptoms were not improved to any great extent, nevertheless the results in the patients' minds seemed to have justified the operation. The results have been classified to draw attention to the important difference between early and late results. The results obtained in the 26 Barnes Hospital cases have been considered separately (Table VII-A). Of the 26 cases definite improvement attributable to phrenicectomy and not to other pre- or postoperative measures was only observed in three cases. There was moderate improvement in five, and slight improvement in four. The results were uncertain in three cases, two were made worse, and six were not improved. The operation was done three times to reduce the size of anatomic defects, that is to diminish the size of cavities which remained after the partial removal of lung tissue with the cautery. The 56 cases collected from the literature have been considered in a similar manner to the Barnes Hospital cases (Table VII-B). The cases from the literature and from the Barnes Hospital have been grouped together (Table VII-C). One can say that 22 (30.55 per cent) showed definite improvement late, nine (12.5 per cent) were moderately improved, nine (12.5 per cent) were slightly improved, and 24 (33.3 per cent) were not improved. Eight (11.1 per cent) were made worse, and the result was uncertain in seven.

In summarizing, therefore, one may say that apparently an individual patient with bronchiectasis has only a relatively small chance of being improved by the operation of phrenicectomy and, on the contrary, a definite chance of being made worse. Moreover, even when improvement occurs it is usually not lasting.

Phrenicectomy as a Prophylaxis Against the Development of Bronchiectasis.—

Phrenicectomy as a prophylaxis against the development of bronchiectasis has been performed on only a very few occasions. We obtained one very striking result in what was considered to be such a possibility. Four other recorded cases (J. A. Moore 2, H. M. Davies 2) show that the results obtained were far from satisfactory. We are very hesitant about even discussing this phase of the subject because of the fear that many patients might be operated upon unnecessarily.

XVIII. LIGATION OF BRANCH OF PULMONARY ARTERY IN THE TREATMENT OF BRONCHIECTASIS

Bruns and Sauerbruch produced carnification of the lung by ligating the branches of the pulmonary artery. And so it was hoped that this procedure might be of value in the treatment of bronchiectasis, particularly by influencing hemoptysis which may be so striking a manifestation of the disease. In 1914, Willy Meyer reported that a branch of the pulmonary artery had been ligated for bronchiectasis on 11 occasions (Sauerbruch, 7; H. Morriston Davis, 1; Willy Meyer, 3). At this time Meyer stated

that all patients were improved but none entirely free of their expectoration. In 1917, he presented a patient whose branch of the pulmonary artery going to the right lower lobe had been ligated 4 years previously (1913). Meyer then stated that his twenty-two-year-old patient had been annoyed previously by frequent expectoration of a large amount of very foul mucopus, but following the ligation of the branch of the pulmonary artery to the diseased lobe expectoration had been reduced from 350 to 15 to 30 c.c. in twenty-four hours. In the above connection one might note that Olch and Ballon were able experimentally to produce abscess of the lung by ligating a branch of the pulmonary artery and incising and suturing the pulmonary parenchyma.

CLOSED ENDOBRONCHIAL ANESTHESIA IN THORACIC SURGERY*

(Preliminary Report)

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WHEN many technics for a given surgical procedure are in vogue, it is usually good evidence that no one is completely satisfactory. This statement applies to anesthesia for intrathoracic surgery.

In 1928, Guedel and Waters[1] described the closed endotracheal technic for administration of inhalation anesthesia. The absorption of carbon dioxide from the anesthetic gases[2, 3, 4] was thus made applicable to surgery of the upper air passages. More recently, the same authors have described further development and application of this technic.[5] There follows a description of a further adaptation of similar equipment to maintain a completely closed endobronchial anesthesia for surgery within the thoracic cage. In their previous communications, Guedel and Waters have described an inflatable balloon or cuff which may be slipped over the various types of to-and-fro endotracheal airways in common use. This inflatable cuff is made of very thin rubber, fits snugly on the outside of the airway and carries a very small inflating tube through which air can be forced into the balloon, bringing its outer wall into air-tight contact with the tracheal wall. By the use of this device, they have found it quite possible to administer anesthesia while the patient's upper air passages are filled with fluid. No leak occurs beyond the inflated balloon. They have anesthetized dogs while both the anesthetic apparatus and the dog were completely immersed in a tank of water.

TECHNIC

To-and-fro endotracheal airways are used ranging in size from 28 French to 34 French for adults. These are usually marketed in fourteen-inch lengths which is ample to reach from the incisor teeth to a point beyond the tracheal bifurcation and well within either bronchus. The inside diameter should be as large as is consistent with reasonable rigidity of the catheter wall. Rubber tubing, silk fabric and silver fabric materials are used in catheters now on the market. The catheter should preferably have a beveled opening in the end. The important qualities are reasonable stability at body temperature in resisting collapse, together with maximum inside diameter in relation to outside diameter.

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The end of such an airway is powdered with talcum and a proper sized inflatable cuff is selected from the stock supply. The cuff is easily brought into position on the catheter if both are well powdered. Now the terminal one and one-half inches of the catheter is moulded in warm water so that it curves toward the side of the bevel tip. The catheter and cuff are sterilized by passing through bichloride solution and sterile water. Sterile vaseline is liberally applied to the airway and the cuff. Anesthesia is induced as for introduction of an endotracheal airway. We have not found preliminary cocainization of the larynx essential. Nitrous oxide or ethylene, with one of the adjuvants ether, tribromethanol or chloroform, when necessary, have been found satisfactory. The vocal cords should be in complete abduction during intubation. This can be assured by proper excess of carbon dioxide in the anesthetic atmosphere just previous to exposure. The glottis is brought into view with a direct vision, anesthetist's laryngoscope and the endobronchial catheter passed rapidly to the bifurcation. The hand carries the catheter close to the right side of the glottis with the curved tip pointing to the left and sliding down the left tracheal wall for entry into the left bronchus and vice versa. After the region of the bifurcation is reached, the airway is advanced slowly into the bronchus, stopping at the first feeling of resistance. Resistance to advance will indicate that the bronchus has been entered too far, reaching a bronchial diameter no greater than that of the airway. Stop short of such resistance if possible. The average distance from cords to bifurcation is eleven to fifteen centimeters. An advance of two or three centimeters beyond the bifurcation is sufficient. The airway is now quickly attached to the anesthesia apparatus. The respiratory space thus consists of one lung, bronchial airway, soda lime canister (to absorb carbon dioxide) and spirometer. This system is filled with anesthetic mixture from the apparatus. A constant flow of oxygen is set to replace that used by the patient in metabolism. The cuff is now inflated with air by means of a ten cubic centimeter glass syringe and the inflating tube sealed with a small forceps. With experience the feel of the plunger in the anesthetist's hand is the best guide as to the proper amount of air to inject. A small water manometer may be used to aid the inexperienced in learning the necessary pressure to insure air-tight closure at the bifurcation without causing undue pressure on the bronchial mucosa. Practice in this regard may be gained by passing the airway into a glass tube and inflating the balloon under direct vision. Anesthesia is then conducted only through the one lung. (See Figure.)

When the chest wall is opened, normal respiration may be allowed to continue or, better, artificial respiration may be instituted. The lung on the side of the operation is allowed to collapse and remain in a state of atelectasis throughout the procedure. Artificial respiration

is under second to second control as to individual depth and minute-volume exchange. A spirometer is used which accomplishes both inflation and deflation of the active lung. A moderately heavy rubber accordion-like bag, cemented to its metal covers, serves the purpose. It may be operated by raising and lowering one cover away from and toward the other by hand or a small motor may be used. A manom-

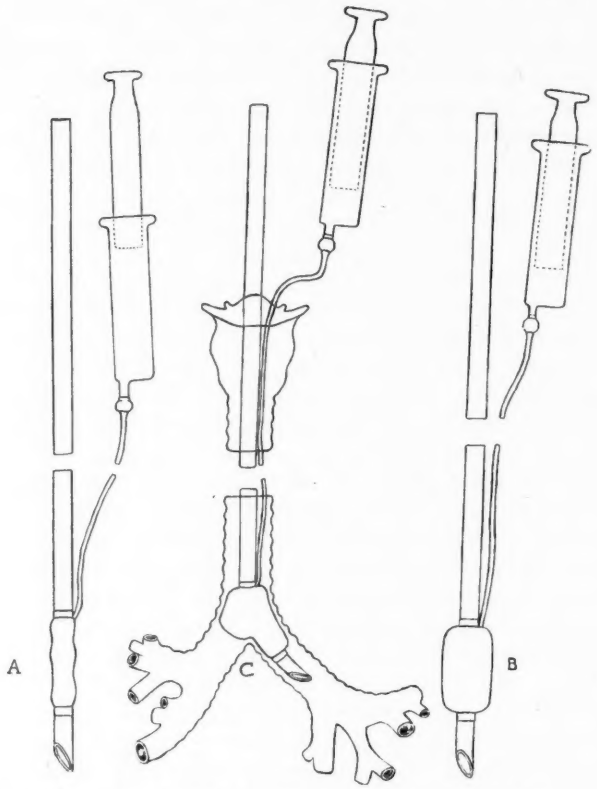

Fig. 1.—(*A*) Shows the catheter with surrounding rubber balloon collapsed. (*B*) Shows the rubber balloon inflated. (*C*) Shows catheter in place and balloon inflated, completely blocking bronchus on the side of operation and insuring non-leak contact of airway in the opposite bronchus.

eter connected to the closed system gives information of the intrapulmonic pressure at all times. At the end of the operation and before closure of the chest cavity, the balloon is deflated, the airway withdrawn into the trachea and the balloon is again inflated. By proper manipulation of pressure in the breathing bag or spirometer, the collapsed lung is again brought into a state of activity by artificial means and inflated to a proper degree to insure filling of the pleural cavity.

After the last stitches are placed in the pleura, sufficient pressure is used to expel completely the air from the pleural cavity, and the closure finished. This pressure, measured in millimeters of water, is maintained throughout the closure of the overlying structures and skin. The surgeon will, of course, make every effort to produce an air-tight closure.

ADVANTAGES

The advantages of a closed endobronchial or one-lung anesthesia for intrathoracic surgery may be considered in two general groups; namely, anesthetic and surgical. By means of closed endobronchial anesthesia and the carbon dioxide absorption technic, it is possible to maintain complete control of anesthetic concentration in a single lung. A warm moist atmosphere is in contact with the lung at all times rather than a cold dry one as is the case with an open system in which moisture and heat must be lost along with carbon dioxide through an exhalation valve. A moderate preanesthetic dose of tribromethanol has been found satisfactory when administered by rectum previous to the induction of inhalation anesthesia. By its use, ether can be completely dispensed with in cases where it is especially contraindicated. By this technic, the control of oxygen and carbon dioxide exchange is directly in the hands of the anesthetist. Anoxemia is eliminated. There is perfect control of artificial respiration as to depth, frequency, and intrapulmonic pressure. Direct observation through the chest wound of the resulting lung activity is a most satisfactory check of the effectiveness of artificial respiration. A manometer to record intrapulmonic pressure gives information as to maintenance of sufficient distention to eliminate pneumothorax during wound closure since the catheter is sealed in place by the inflation of the rubber balloon.

The advantages offered the surgeon through this method of anesthesia are worthy of consideration. Intrathoracic procedures for the thorough exploration of the lung or removal of a lobe necessitates a large opening in the chest wall. Because of the danger resulting from such wide thoracotomy wounds, surgeons in many instances have been willing to operate with inadequate exposure through a small opening with disappointing results. The sudden creation of an open pneumothorax with a resultant collapse of the homolateral lung and a shifting of the mediastinum to the side of the contralateral lung produces a great diminution in the patient's vital capacity which causes considerable shock. To this is added paradoxical respiration with inadequate oxygenation of the blood and irregular and violent respiratory movements. The returning blood flow to the heart is immediately altered in quantity and regularity. This initiates a sudden circulatory disturbance which when combined with the radical alteration in respiration may bring about sudden and sometimes fatal circulatory col-

lapse. If the patient successfully endures the early effects of such procedure and the respiratory movements are held down to a minimum through careful anesthesia, his chances of surviving the operation are much greater. The continuation of such conditions for more than a few minutes, however, materially decreases the chances for recovery.

With the method above described, the one lung is allowed to function under slightly more than atmospheric pressure. This is sufficient to prevent the raised pressure in the open pleural cavity from pushing the mediastinum against the contralateral lung, thereby diminishing the vital capacity to that extent. With this technic and artificial respiration, the lung which is receiving the anesthetic gases is permitted to maintain its respiration to practically a normal degree. At the same time, all paradoxical respiration is eliminated. The functioning lung is given a sufficient amount of oxygen to prevent oxygen want. The volume of blood passing to the heart is held constant and sudden circulatory collapse is not encountered. The blood pressure and pulse rate remain constant. The only movement observed on the side of the operation is that due to the pulsation of the great vessels. The lung is collapsed and immobile. The lobes remain pink throughout the operation. Exposure is excellent and the procedure may be carried on without interruption. Ligatures and sutures can be deliberately applied. Dislodgment of infected material is reduced to a minimum because of the advantageous deflation of the lung. Following the ligation of the pedicle, the balloon around the catheter is partially deflated, withdrawn into the trachea and reinflated. The operated lung is then gradually inflated. If a leak or fresh bleeding occurs, the balloon is immediately deflated, reinserted into the bronchus to effect immobility of the operated side, and the repair made at once. The operated lung is again gradually and completely inflated while the pleura is being closed. Just before the final closure, the lung is well distended to eliminate pneumothorax and maintained in that condition until the dressing is in place. The whole procedure can be carried out very rapidly without undue shock to the patient and without any radical or sudden pressure changes occurring during the entire time.

SUMMARY

Closed endobronchial anesthesia has been employed in several experimental animals for the removal of different lobes of the lung, and in one patient for the removal of a mediastinal tumor. Artificial respiration may be maintained in the one lung with perfect control of its respiration and minute-volume respiratory exchange. Intrapulmonary pressure is measured by a side tube to a manometer. The incorporation of the closed carbon dioxide absorption technic assures a warm moist anesthetic atmosphere at all times.

The shock usually encountered upon the sudden creation of an open pneumothorax has been very slight. This has been demonstrated by the blood pressure and pulse rate remaining constant. The irregular and violent respiratory movements, so often seen, have been replaced by slow, regular and deep breathing. Exposure is simple for the surgeon because the lung in the operative field is immobile. Chances of infection are reduced because a sucking wound has been entirely eliminated. Little chance exists for infected secretions to be squeezed into the trachea because the bronchus is blocked.

Although the method has been employed in a series of cases, too small from which to draw conclusions, the evidence so far obtained gives promise that the closed endobronchial type of anesthesia is a most practical method to employ in intrathoracic operations in which the pleural cavity must be opened.

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MULTIPOSITION BED FOR CONTINUOUS POSTURAL DRAINAGE*

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THIS bed† has been developed because the need has been shown for continuous drainage in lung abscess, bronchiectasis and other suppurative lung conditions. The purpose was to have not only a bed which looked like other beds in the wards but also one that could be easily manipulated by a nurse or attendant. In postoperative care in all surgical conditions the moving of the patient from side to side has always been difficult. However, by turning the proper crank on this bed one can shift the patient from side to side or up or down with very little effort. The bed meets requirements of the standard hospital bed; it stands twenty-eight inches from the floor, which includes four-inch rubber tire rollers. The foot of the bed permits elevation of twenty-three inches—an angle of 35 degrees. The side to side tilt of the springs is ten inches—an angle of 40 degrees. Side guards are furnished with the bed and are to be employed when lateral positions are used. The tilt of the spring frame is accomplished by means of the crank handle which is situated in the lower left hand corner of the bed. Two other crank handles are at the foot of the bed, one for elevating the entire frame at the foot end, and the other for elevating the back rest of the head. All of these handles can be turned under the framework while not in use.

Fig. A shows a patient lying comfortably in bed with the head rest up. The bed as a whole looks like any standard bed in a hospital. Fig. B shows the foot of the bed raised to an angle of 35 degrees. The patient at this time is lying on her back but could be turned anteriorly; this position is excellent for patients to be rolled after operation to prevent shock. The arrow points to the side brackets which are removable when not in use. Fig. C shows the patient with head down and to the left. If one desires to turn the patient in the opposite direction the lower crank handle is turned which reverses the entire position.

The advantages of this bed are as follows:

1. Almost any desired position for use in postural drainage is obtained by very little effort on the part of the operator and no effort at all on the part of the patient.

2. It permits one to carry out either intermittent or continuous postural drainage.

3. Regular standard bed is used with the fittings below, which make them inexpensive.

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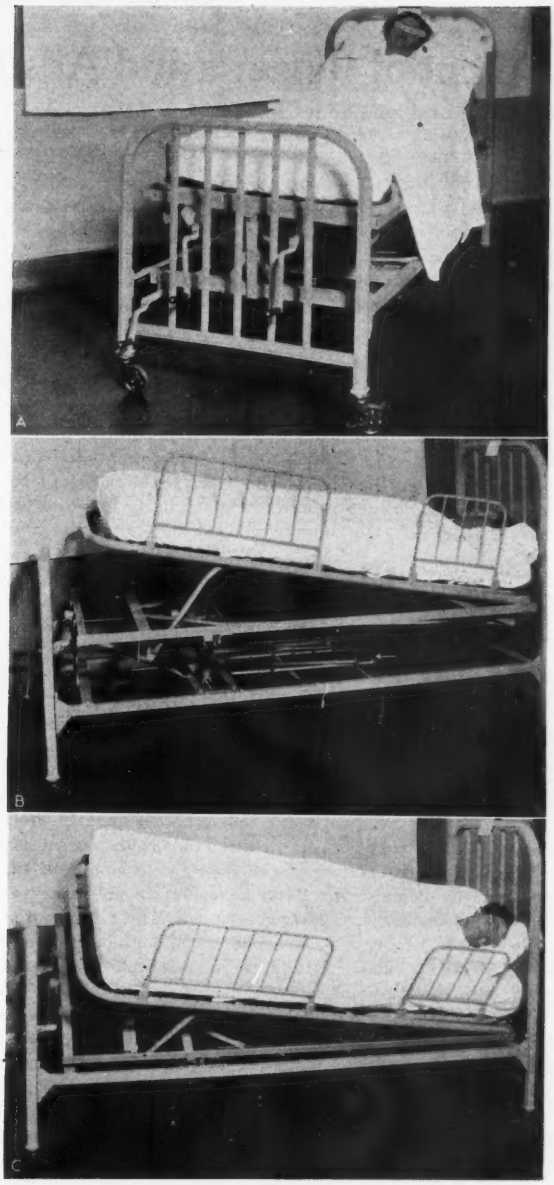
†Made by Smith & Davis Mfg. Co., St. Louis, Mo.



Multiposition bed for continuous postural drainage. *A, B, C* show actual positions used in treating a case of lung abscess. (See text.)

Department of Reviews and Abstracts

Conducted by Duff S. Allen, M.D., Associate Editor

Selected Abstracts

Kazda, F., and Stöhr, W.: Zur Frage der tödlichen Lungenembolie. (The Question of Fatal Pulmonary Emboli.) Deutsche Ztschr. f. Chir. **231**: 187-226, 1931.

The authors present the results of a study of the aggregate incidence of fatal pulmonary emboli from the material of the Pathological Institute of Vienna and offer the conclusion that fatal pulmonary emboli are more common in nonsurgical cases than in cases surgically treated. Two periods of time were studied, (1) 1915-1918 and (2) 1922-1928. The total number of fatal postoperative pulmonary emboli of the combined reports amounted to 145. Those from internal diseases alone in the second seven-year period amounted to 152. From the statistics obtained, it is to be noted that middle-aged women are particularly prone to postoperative fatal pulmonary emboli, but that on the other hand during the war years an unusually high proportion was spared. It is suggested that the lessened degree of adiposity during the war years may have some relation to this lowered incidence of fatal postoperative pulmonary emboli. It is furthermore important that in over half the cases of postoperative fatal pulmonary emboli, lesions of the heart were found. In slightly less than 50 per cent of the cases there were lesions of the respiratory tract. In about 33 per cent of cases changes in the spleen were present. The conclusion seems possible that operative trauma as the etiology for postoperative pulmonary emboli can be considered but superimposed upon a fundamental metabolic substratum and accompanying organic lesions. This hypothesis is offered for the well-known phenomenon of the localization of the thrombosis far from the operative site, usually in the right lower extremity. Fatal pulmonary emboli in the course of nonoperated disease did not show the regression in 1928 that the postoperative pulmonary emboli demonstrated. However, it is probable that clinicians are more conversant with the condition and are more prone to diagnose it. Fatal nonoperative pulmonary emboli have a pronounced seasonal incidence, occurring most frequently during the inclement weather periods (April, October and November). In consideration of the accompanying diseases, it is particularly noteworthy that organic lesions of the central nervous system occur in quite as high a percentage as cardiac lesions, that distant lesions of the respiratory tract occur in 66 per cent of cases, that lesions of the liver and biliary passages are present in more than 50 per cent and that lesions of the heart and vessels are present in only about 50 per cent. Nonoperative fatal pulmonary emboli seem more dependent upon the accompanying lesions in the viscera, other than those the site of the disease most in the foreground, than operative cases. It is noteworthy that the greatest number of postoperative fatal pulmonary emboli were coincident with the grippe epidemic of 1918. The authors do not ascribe much importance to the rôle played by wound infection, intravenous therapy or local anesthesia technic. The most important factor to the authors is a fundamental metabolic and organic diathesis, morbid obesity.

Felsenreich, Fritz: Konservative oder operative Therapie Dei Vorhofs verletzungen des Herzenes. (Conservative or Operative Therapy in Injuries of the Cardiac Auricles.) Arch. f. klin. Chir. 166: 4, 1931.

The development of surgical diagnosis and technic has brought surgery to the point of abandoning the conservative management of penetrating injuries of the heart and giving the greatest possible consideration for the operation of cardiac suture. Forty-four per cent of operative cures stands as a vivid contrast to 12 per cent good end-results with nonoperative treatment. Undoubtedly with the aid of the already published great number of operatively cured cases the appropriateness of operative measures can be ably demonstrated. On the II Surgical Clinic in Vienna the standpoint has been taken of immediate operation upon the certain diagnosis of perforatory wounds of the heart. They have procrastinated only in some of the very recently observed cases of certain penetrating wounds of the heart when through other suitable conservative measures they have been able to recover these patients. Nevertheless these were rare and unusual cases and conservative management was straightway abandoned at the first appearance of menacing symptoms and operative treatment was instituted.

An imperative necessity for operation does not hold in the absence of signs of progressive cardiac tamponade or severe anemia. However the following considerations speak for immediate operation: (1) The danger of secondary hemorrhage into the pleura or pericardium with consequent exsanguination or menacing cardiac tamponade. (2) Thrombus formation along the line of the site of the wound of the cardiac wall with consequent embolic accidents.

Against the performance of an operation are the following points: (1) The very precarious operative prognosis of wounds of the auricles. (2) The rapid improvement in the general condition with morphinization. (3) The absence of the appearance of progressive cardiac tamponade. (4) The dangers accompanying free exposure of the right auricle (resection of sternum). (5) The late complications of operation, notably infection, the incidence of which is estimated at 30 per cent in the combined statistics of all authors.

Cases of auricular injuries observed by the author healed by nonoperative treatment lead him to plead for an evaluation and treatment of injuries of the auricle differing from that of injuries of the ventricle. The distinction is based principally on the differences of the internal pressures of the auricular and ventricular cardiac chambers. Upon theoretical considerations the third stage of life-endangering cardiac tamponade cannot be attained in injuries of the auricle because of the equalization that occurs between the internal and external cardiac pressures.

The use of morphinization as a differential diagnostic measure to ascertain whether the shock is due to bleeding into the pleural or pericardial sacs is stressed. The shock due to injury or hemorrhage into the pleural sac will respond to morphinization whereas the lowered blood pressure and shocked appearance of progressive cardiac tamponade will not be alleviated. Pericardial puncture has not served as a useful therapeutic measure, in the sense of a lasting relief of the increased intrapericardial pressure, but rather has served as a diagnostic measure in dubious cases to differentiate injuries of the right or left heart and to estimate the existing degree of intrapericardial pressure.

In conclusion the author states that (1) intervention is always justified in cardiac injuries in the presence of evidences of severe anemia and cardiac tamponade. (2) Most cardiac penetrating injuries occur through wounds of the left chest encountering either the left or right ventricle. These demand immediate operation in contrast to wounds of the base of the heart, especially those of the right auricle, which are proportionately trifling and less menacing to the life of the patient. Operative attack for this type of injury may be technically very difficult and very

dangerous. (3) If the indications for immediate operation are absent nevertheless it must be borne in mind that a ventricular injury may be present and at the first presentation of a suggestive symptom of ventricular injury prompt exploratory exposure of the heart is indicated. (4) In those patients with injuries to the base of the heart in whom the considerations of (a) a fairly good prognosis, (b) of the gravity of resection of the sternum for exposure, and (c) the difficulty of suture of the vena cava and right auricle speak against operation, conservative measures should be employed initially and operation delayed.

Schlomka, G.: Elektrokardiographische Beobachtungen bei Herzstichverletzung. Beitrag zur Genese der Angina pectoris. (Electrocardiographic Observations in a Case of Cardiac Wound. Note on the Genesis of Angina Pectoris.) Deutsche med. Wchnschr. **57:** 630, 1931.

The author observed a case of severe penetrating wound of the heart and conducted a continuous electrocardiographic study upon it. The electrocardiogram revealed changes referable to severe acute coronary artery closure. This stands in contrast to the well known occasional failure of clinical symptoms to diagnose acute coronary artery closure. The electrocardiographic study was able to diagnose a widespread acute closure of a coronary artery and to detail the sequelae of it. All of the familiar painful anginoid syndrome was completely absent. The author states therefore that coronary closure causing heart muscle ischemia may not necessarily be the basis of the painful anginoid syndrome. The electrocardiographic diagnosis of coronary artery injury was fully confirmed by postmortem examination.

Bijlsma, U. G., and le Heux, J. W.: Die Bedeutung des Perikards für die Dynamik des Säugetierherzens (The Significance of the Pericardium in the Dynamics of the Cat's Heart). Arch. f. exper. Path. u. Pharmakol. **159:** 12, 1931.

Up to a certain degree dilatation of the heart favors an increase in cardiac action. This degree of dilatation is named the "physiological dilatation end point" by the authors, (called by Starling "the optimal volume"). This investigation proposed to answer the questions: (1) whether the influence of the pericardium normally permits the attainment of this "physiological dilatation end point," (2) whether, by its eventual hindrance of dilatation through its enclosing tension when stretched, the pericardium ever allows maximum efficiency to be obtained. Investigations were undertaken on heart-lung preparations of the cat with observations of the minute volume output and intraventricular pressures. With this preparation it was possible by aid of suture closures of the pericardium to demonstrate variations in output with the opened and closed pericardium. The investigations demonstrated changes (1) due to aortic closure, (2) due to the high peripheral resistance in the arterial tree, (3) through increase in the back pressures particularly of the right heart. Under the first condition the pericardium demonstrates its action when present through a higher initial cardiac efficiency. Under the second condition of the increased peripheral pressures, the minute volume progressively decreased when the pericardium remained intact until standstill occurred; however, the heart freed of its pericardium under similar conditions ceased activity at a much earlier time. Under the third condition the minute volume was much greater with pericardium intact than in hearts freed of pericardium. The conclusions drawn are:

(1) The pericardium serves as a protective measure against excess dilatation of the heart. (2) Dilatation of the heart in the presence of an intact pericardium to the "physiological dilatation end point" is impossible, and the heart cannot draw upon it as a measure of physiological reserve. For this "waste" by the heart with

intact pericardium there are substituted the compensatory reserve power of increase in function effected after rises in diastolic pressure and the greater efficiency of cardiac action in the presence of initially high peripheral pressures which are exhibited only by the heart with an intact pericardium.

Morian, R.: Zur traumatischen Spieseröhrenlahmung. (Traumatic Esophageal Paralysis.) Arch. f. klin. Chir. **164:** 226-232, 1931.

This report is noteworthy because of the rarity of reports on the nerve centers for salivary secretion. In a case of unilateral fracture of the base of the skull, the IX, X, XI and XII nerves were compressed by the bony edges of the fracture line. There occurred a complete paralysis of the pharynx and esophagus and, as a result therefrom, a fatal aspiration pneumonia. There was, moreover, a paralysis of the recurrent laryngeal and facial nerves. In the nucleus of the glossopharyngeus, immediately below the inferior olive, somewhat internal to the facial nucleus, degenerated cells were found. The locality corresponds accurately to the primary cells for salivary secretion as have been previously described. Clinically no noteworthy demonstration of this physiologic unit is usually available because of the compensating ability of the opposite side. Distinctly fatal results from unilateral traumatism of the vagus may be manifested through the effects of the complete paralysis of the pharynx and esophagus without paralytic involvement of the heart and lungs.

Pirker, H.: Ueber Behandlung von Esophagusverengerungen. (Treatment of Esophageal Strictures.) Arztl. Praxis **5:** 135, 1931.

Gastrostomy is indicated when, because of an inflammatory swelling or fibromuscular contracture, a more or less complete stenosis of the esophagus occurs, or when a periesophageal abscess appears. In the treatment of caustic erosions of the esophagus early bouginage is employed, which, following the proposal of Salzer, is begun from two to six days after the subsidence of the height of the inflammatory picture. The bougie used usually is an elastic, red rubber, solid sound No. 30. In the severe cases that exhibit the vomiting of blood, tissue and necrotic debris one should wait two to three weeks before beginning bougie treatment. Only exceptionally at the initial bouginage can large bougies be passed. The bougies are coated preliminarily with an anesthetic paste. Highly sensitive patients first swallow a teaspoonful of 5 per cent solution of anesthesin chloride. In cases of tight strictures filiform bougies are introduced along swallowed conducting axis threads of catgut. If the usual bouginage by mouth does not succeed, esophagoscopic manipulation is performed. In the presence of a gastrostomy, retrograde bouginage is recommended, particularly if bouginage from above does not meet with complete success. Electrolysis with the galvanic current using from 2-5 ma. is useful for apparently unpermeable strictures. When one cannot gain his objective with these means, the following chemical aid may be of service. Doses of ferric lactate solution are taken. After a short lapse of time the gastric juice passes upward and forms a dark ferric acid salt which clearly outlines the lumen of the otherwise invisible aperture of the stricture and thus facilitates the esophagoscopic passage of filiform bougies through it. When all these attempts fail a plastic operation upon the esophagus is to be undertaken. In cases of cardiospasm dilatation must be employed until a circumference of 10 to 12 cm. is attained. For this purpose both the sound of Gottstein or the instrument of Von Brünings are recommended. According to Lothheisen's experience success is usually attained with this procedure.

Schoen, R.: Untersuchungen über Kiefer—und Zungenreflexe. III Mitteilung: Die Beziehungen von Zunge und Kiefer zur Atmung und ihre Bedeutung fur die Kenntnis der zerebralen koordination der Atmung. (Researches on Jaw and Tongue Reflexes—III Report: The Relation of Jaw and Tongue to Respiration and Their Importance in the Recognition of the Cerebral Coordination of Respiration.) Naunyn-Schmiedeberg Arch. f. Exp. Path. und Pharm. **161:** 4-5 August 1931, p. 573.

The authors have previously described their method of graphic analysis of the association of tongue and jaw movements in cats with that of the associated muscles after extirpation of the cerebrum, the thalamus or midbrain. The associated respiratory movements were registered on a Marey capsule attached to a tracheal cannula. Frequently the movements of the larynx likewise were demonstrated. The same results were noted in rabbits. With the onset of asphyxia respirations were continued with the use of CO_2 admixture. The cerebral manipulations were controlled later by autopsy. Morphine and pernoctan were used in many experiments to produce respiratory disorders.

A graphic analysis of the association of tongue, jaw and laryngeal movements of cats with the associated reflex respiratory movements demonstrates definitive basic types of movement coordination. With the apnea reflex (the first phase of deglutition, inhibited inspiration) there occurs with the cessation of respiration closure of the mouth and retraction of the tongue. With the inspiratory reflex (stimulation of the nasal orifices, the first phase of the sneezing reflex), inhalation is favored through immediate opening of the mouth, retraction of the tongue and rise of the larynx. The expiratory reflex (defense reflex, coughing, sneezing) is for the most part combined with the apnea reflex.

Likewise the same mechanism operates in spontaneous interruptions of respiration. A notable example occurs in asthmatic respiration in which type a single deep sigh when ingrafted upon otherwise normal respiration can be the precipitating sign of this disorder of respiration. This stands out as an inspiratory reflex of the highest grade. It can be induced through peripheral stimuli. In choking respiration and emesis convulsive expiratory movements can be demonstrated. The apnea reflex has its analogue in periodic respiration.

The charted curves of associated respiratory movements of expiration appear to bear quite a similarity to the respiratory reflexes of asthma with its subsequent assumption of normal respiration. Analogies are presented to other well known forms of respiratory disorder. It seems possible to analyze the well known clinical types of respiration in terms of types of associated respiratory movements and arrive at the conception of a central coordination of respiration.

Murray, D. W. G.: Method of Palliative Treatment of Carcinoma of the Esophagus. Canad. M. A. J. **25:** 271, 1931.

Gastrostomy or enterostomy to prevent starvation does not relieve the distressing periodic regurgitation of mucus. Intermittent dilatation requires repetition at short intervals. Dilatation of the stricture and intubation of the lumen as introduced by Simmons and Soutar eliminates the symptoms of obstruction and regurgitation of mucus and obviates the necessity of a gastrostomy which restricts the patient from society generally.

A bougie guide is introduced initially under esophagoscopic guidance using a Cameron lighting system. The tubes, composed of coil spring wire (alloy of silver and nickel), can be accommodated to any distortion while retaining patency. They are passed along the line of the bougie. Sixty per cent of esophageal carcinomata occur about the level of the tracheal bifurcation. These are usually of the squamous cell type, fairly firm and not very vascular. All these conditions are suitable for the

introduction and retention of the tube. In the lower third of the esophagus, comprising about 30 per cent, the growths tend to be more vascular and softer in consistency with a greater tendency for the tubes to drop through. Hypopharyngeal and postericoid growths are unsuitable. Cardia growths are less satisfactory because of constant gastric regurgitation.

Ten cases so treated are reported. No difficulty in placement of the tube as desired was encountered. In none was hemorrhage or infection produced at the operation. It provided relief from obstructive symptoms for periods up to ten months in this series of cases. Some of the patients returned to work.

Beattie, J.: Importance of Anomalies of the Superior Vena Cava in Man. Canad. M. A. J. 25: 281, 1931.

The presence of anomalies of the great venous trunks in the upper thorax must be considered when a diagnosis of aortic enlargement is made from radiograms of this region. In radiograms of the thorax the superior vena cava is visible in almost every case after the thirty-fifth year forming a part of the "aortic" or "vascular" shadow in frontal and oblique radiograms. In oblique views the posterior edge of the so-called "aortic shadow" is always formed by the vena cava superior or the vena cava may project somewhat behind it.

Anomalies of the superior vena cava can be divided into three classes, (1) persistence of both primitive anterior cardinal veins and the normal cross anastomosis between them, (2) persistence of both primitive anterior cardinal veins but with no cross-anastomosis between them, (3) persistence of the left anterior cardinal vein with obliteration of the right. Persistence of the anterior cardinal vein with obliteration of the left is the normal arrangement. Thus far 175 cases of anomalies of the superior vena cava have been recorded in the literature. Types I and II far outnumber type III. The anomalies are not incompatible with life. Three specimens are described in this report found in autopsies performed on adults.

Knutsson, Folke: On Intrathoracic Neurinomata. Acta radiol. xii. f. 30. ix. 1931.

Intrathoracic neurinomata originating from the roots of the thoracic nerves have an hour-glass shape with an intraspinal and paravertebral portion. The latter part may extend along the spinal nerve for some distance into the intercostal space or be located in the posterior part of the mediastinum beside the spinal column. This part appears on the radiogram as a massive, well-defined, more or less founded tumor shadow which occupies quite a typical position in the thorax. The tumor is thus always localized to the posterior part of the thoracic cavity either intercostally or paravertebrally. It is always located extra-pleurally and on the radiogram it is usually possible to see how the pleura from the chest wall is reflected over it. This localization serves to differentiate the neurogenic tumor not only from other mediastinal tumors which are located more anteriorly but also from accessory goiters, thymic tumors, mediastinal cysts, and aneurysms of the ascending arm and arch of the aorta. Their position coincides with aneurysm of the descending aorta, but a differential diagnosis is here possible on other grounds. The differential diagnosis may be impossible in rare cases by confusion from fibroma, sarcoma or tumors of that nature arising from the connective tissue of the posterior mediastinum. If there are signs of the tumor being of hour-glass shape this is an additional factor to favor neurinoma. In support of an hour-glass tumor will be spinal cord pressure symptoms, signs of occlusion of the subarachnoid space by myelography and enlargement of the inter-vertebral foramina roentgenologically. In some cases an artificial pneumothorax will be of service to demonstrate that that tumor is nonpulmonary in origin.

The neurinomata grow fairly slowly and in the majority of cases have symptoms dating back for several years. They occur at all ages. They generally give rise to neuralgic pains which obtain a segmental localization. They may grow periodically at varying speeds. True neurinomata scarcely ever undergo malignant degeneration in contrast to neurofibromata which not infrequently undergo sarcomatous transformation. Three cases of neurinomata are described, of which two were verified by operation and pathologic examination while in the third case the diagnosis could only be made probably on the grounds of clinical data and the roentgenogram.

Walzel, P.: Zur Klinik und Therapie der Mediastinaltumoren neurogener Abstammung. (Clinical Aspects and Therapy of Neurogenic Mediastinal Tumors.) Arch. f. klin. Chir. **163:** 626-639, 1931.

The author presents a case of neurogenic posterior mediastinal tumor which, slowly growing, attained enormous size. It weighed 1¾ kg. and was 20 by 15 cm. in diameter. It arose on the right side of the mediastinum and grew outwards into the fissure between the middle and lower lobes of the right lung. The right lower lobe was compressed thereby and rendered atelectatic. At operation, wide exposure of the tumor revealed it to be grossly adherent to the contiguous mediastinal structures. During the course of the extirpation the right vagus nerve was in all probability irritated, the consequences of which were severe meteorism and altered intestinal function for many weeks postoperatively. The large operative wound defect was filled in with tampon. Convalescence was smooth. A secondary operation for closure of the operative wound defect was necessary. Microscopic study of the tumor revealed it to be a neurinoma. The author suggests that such cases should have primary closure of the operative wound in spite of widespread oozing to avoid the necessity of a secondary operation.

Steinfeld, E.: Bronchomycosis. M. Clin. North America, **15:** No. 2, 403, September, 1931.

The division of the mycoses into severe and mild has significance in discussion of bronchomycosis as it is found in this country. The severe forms are usually due to coccidioidal granuloma, blastomycosis (oidiomycosis), nocardiosis and torula infection. The mild types are as a rule due to yeasts of the genus Monilia or related genera even though these yeasts may create severe infections similar to those described in the Tropics.

In severe pulmonary mycosis, radiograms may show evidence of infiltration and actual nodules. In mild cases these may be simply peribronchial thickening and fibrosis. No distinctive picture has been described. Yeasts of the genus Monilia particularly may cause various respiratory diseases such as chronic bronchitis, asthmatic bronchitis and conditions resembling tuberculosis. Severe mycoses are usually due to Torula (which differs from other mycoses in that it is occasionally complicated by cerebral invasion), Oidium, Coccidiodes and Nocardia. Fungus sensitivity causing bronchial asthma may be due to molds such as aspergillus, Alternaria, Mucor and others. These may cause bronchial and pulmonary infection. The presence of yeasts in the sputum does not of itself indicate any pathologic significance. The presence of large numbers of colonies particularly in the vegetative form disappearing concomitant with clinical improvement is significant. Inoculation into susceptible animals is of value when definite pathologic lesions can be produced.

It is important to clear up upper respiratory focal areas which act as reservoirs of infection. In fungus sensitivity the use of a vaccine prepared from the offending fungus may be useful. Iodides in large doses are usually effectual in mild cases of bronchomycosis.

Book Review

MATAS BIRTHDAY VOLUME, A COLLECTION OF SURGICAL ESSAYS written in honor of Rudolph Matas, New Orleans, Paul B. Hoeber, Inc., New York, 1931.

It is particularly fitting that the distinguished career of Rudolph Matas should have been memorialized by this collection of essays from pupils and friendly admirers. The influence of Dr. Matas as shown in this collection of essays was a considerable one in the field of Thoracic Surgery. In this collection there appear, however, only two articles which properly belong to this domain, one is by Lilienthal on the surgeon's moral obligation in the operative treatment of phthisis. The other is by Graham describing observations on the reaction of bronchial fistulae to acute infections of the upper respiratory tract. Other contributors to the volume are Mont R. Reid of Cincinnati, Leriche of Strasbourg, Alessandri of Rome, Royster of Raleigh, Bloodgood of Baltimore, the Finneys of Baltimore, Coffey of Portland, Ribas of Barcelona, Hoffman of Newark, Horsley of Richmond, the late John B. Deaver of Philadelphia, Crile of Cleveland, Moure of Paris, Trias-Pujol of Barcelona, W. J. Mayo of Rochester and D. P. D. Wilkie of Edinburgh. The book as a whole is a splendid tribute to a distinguished member of the Editorial Board of this Journal and there is much interesting reading within it. The work is well illustrated and the general appearance is excellent.

Erratum

In the title of the article by Brill, Prinzmetal, and Brunn, page 243 of the February issue of the Journal, the second word, Alternating, should read *Altering*—Factors Altering Intrapleural Pressure and Their Clinical Significance.

The American Association for Thoracic Surgery

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The American Association for Thoracic Surgery

Ann Arbor, Michigan, April 18, 19 and 20, 1932

Program

Monday, April 18

9:00 a. m.—Clinical Demonstrations and Discussions.
By staff of University of Michigan Hospital.

1. "The Evaluation of the Cardiocirculatory Risk in Thoracic Surgery."
   Dr. Paul Barker.
2. "Response of Primary and Secondary Pulmonary Neoplasms to Radiation."
   Dr. Harold W. Jacox.
3. "Utility of Lateral Roentgenograms in the Diagnosis of Obscure Thoracic Conditions."
   Dr. Carleton Peirce.
4. "Anomalies of the Diaphragm."
   Dr. F. J. Hodges.
5. "Dehydration Attendant Upon Surgical Operations."
   Dr. F. A. Coller and Dr. Walter G. Maddock.
6. Presentation of Nontuberculous Thoracic Surgical Patients by
   Drs. John Alexander, Cameron Haight, Duane Carr and
   J. Dewey Bisgard.

1:00 p. m.—Luncheon at the University Hospital.

2:00 p. m.—Scientific Meeting, University Hospital.

1. Business Meeting of the Association.
2. "The Surgical Treatment of Carcinoma of the Lung."
   Dr. Edward D. Churchill, Boston.

3. "Putrid Lung Abscess. Its Pathogenesis, Pathology, Symptomatology, Diagnosis, Surgical Treatment and Results."
   Dr. Harold Neuhof, New York City.
4. "The Medical Treatment of Abscess of the Lung."
   Dr. Conrad Georg, Jr., Ann Arbor.
5. "Some Observations Upon the Value of Bronchoscopy in the Treatment of Bronchiectasis."
   Dr. David H. Ballon, Montreal, Canada.
6. "The Inseparability of Endoscopy and Thoracic Surgery."
   Dr. William A. Hudson, Detroit.
7. "Concerning the Nature and Function of Certain Afferent Pathways From the Thoracic Viscera."
   Dr. Peter Heinbecker, St. Louis.
8. "A Case of Parasitic Fetus of the Anterior Mediastinum."
   Dr. Stuart W. Harrington, Rochester, Minnesota.
9. "The Nature, Prevention and Treatment of Postoperative Pulmonary Complications in Chest Surgery."
   Dr. Pol N. Coryllos, New York City.

8:30 p. m.—Reception for Members and Their Guests.
Dr. John Alexander and Dr. Conrad Georg, Jr.
2121 Wallingford Road.

Tuesday, April 19

9:00 a. m.—Clinical Demonstrations and Discussions, University Hospital.

1. "Presentation of Roentgenograms and Results of Extrapleural Pneumolysis With Paraffin Filling."
   Dr. M. C. Indoe.
2. "Presentation of Roentgenograms and Results of Cauterization of Pleural Adhesions Under Thoracoscopic Control."
   Dr. Anderson.
3. "Management of Poor Surgical Risks for Thoracoplasty in Tuberculosis."
   Dr. E. J. O'Brien.
4. "The Value of Temporary Phrenic Paralysis in Tuberculosis."
   Dr. John B. Barnwell.
5. Presentation of Tuberculous Thoracic Surgical Patients by
   Drs. Alexander, Barnwell, Haight, Carr, and Bisgard.

1:00 p. m.—Luncheon at the University Hospital.

2:00 p. m.—Scientific Meeting, University Hospital.

1. Executive Meeting of the Association.
2. President's Address.
3. "Special Considerations With Regard to Closure of Large Upper Lobe Tuberculous Cavities."
   Dr. John Alexander, Ann Arbor.
4. "Cautery Pneumolysis for Pulmonary Tuberculosis."
   Dr. E. S. Welles, Saranac Lake.
5. "The Suspended Cavity in Artificial Pneumothorax."
   Dr. E. J. O'Brien, Detroit.
6. "The Treatment of Tuberculous Empyema."
   Dr. Carl A. Hedblom, Chicago.
7. "Tuberculous Pyopneumothorax With Infection."
   Dr. Frank E. Berry, New York City.
8. "Bilateral Partial Thoracoplasty for Bilateral Pulmonary Tuberculosis."
   Dr. Duff S. Allen, St. Louis.

9. "The Present Status of 100 Patients Treated With Artificial Pneumothorax After One to Eighteen Years' Reexpansion of the Lung."
Dr. Edward N. Packard, Saranac Lake.

6:30 p. m.—Reception for Association.
Dr. Frederick A. Coller, 2105 Wallingford Road.

8:30 p. m.—President's Banquet, Michigan Union.

Wednesday, April 20

9:00 a. m.—Scientific Meeting, University Hospital.

1. "A Study of Phrenicectomy in Pulmonary Tuberculosis."
Drs. Lawrason Brown, J. N. Hayes (by invitation) and J. T. Eagan (by invitation), Saranac Lake.
2. "Thoracoplasty in Tuberculosis of the Lung."
Dr. Adrian V. Lambert, New York City.
3. "Surgical Immobilization Therapy in Tuberculosis."
Drs. Evarts A. Graham, J. J. Singer, Duff S. Allen, and Harry C. Ballon, St. Louis.
4. "Eight Years of Selective Collapse Surgery for Pulmonary Tuberculosis."
Drs. Frank S. Johns and Dean B. Cole, Richmond.
5. "Extrapleural Thoracoplasty in the Presence of a Closed Pneumothorax or Hydrothorax."
Dr. Ralph B. Bettman, Chicago.
6. "Experiences With Carcinoma of the Esophagus."
Dr. Carl Eggers, New York City.
7. "Actinomycosis of the Thorax With Report of a Case Successfully Operated Upon."
Dr. Owen H. Wangensteen, Minneapolis.
8. "The Treatment of Chronic Empyema."
Dr. Deryl Hart, Durham, North Carolina.
9. "The Thoracic Complications of Subphrenic Infections."
Dr. Harold Bye, Iowa City.

For those who wish to plan to remain in Michigan after the formal conclusion of the Association program, the following operative clinics have been arranged:

Dr. John Alexander, Thursday, Friday and Saturday mornings at the University of Michigan Hospital.

Dr. E. J. O'Brien, Thursday and Friday mornings at the Herman Kiefer Hospital, Detroit. Saturday morning at the American Legion Hospital, Battle Creek.

Dr. William Hudson, Thursday morning at the Receiving Hospital, Detroit. Friday morning at the Grace Hospital, Detroit. Saturday morning, place to be announced during the meeting.